

**The social predictors, psychological and affective processes  
of formal thought disorder in patients diagnosed with  
schizophrenia-spectrum disorders**

Thesis submitted in accordance with the requirements of the  
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**DECLARATION IN HIGHER DEGREE THESES**

**DECLARATION**

This thesis is the result of my own work. The material contained in the thesis has not been presented, nor is currently being presented, either wholly or in part for any other degree or qualification.

Signed ..... *Paulo Sousa* .....(candidate)

Date .....09/06/2015.....(candidate)

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# Table of contents

<b>Acknowledgments</b>	<b>2</b>
<b>Table of contents</b>	<b>4</b>
<b>List of tables</b>	<b>12</b>
<b>List of figures</b>	<b>14</b>
<b>List of appendices</b>	<b>15</b>
<b>Chapter 1. Introduction</b>	<b>16</b>
<i>1.1 Introduction</i>	<i>17</i>
<i>1.2 References</i>	<i>22</i>
<b>Chapter 2. The psychology of thought disorder: A narrative review</b>	<b>28</b>
<i>2.1 Introduction</i>	<i>29</i>
<i>2.2 The nature of thought disorder</i>	<i>31</i>
2.2.1 What is so disordered about thought disorder?	31
2.2.2 TD as a heterogeneous construct	36
2.2.3 What is so “schizophrenic” about thought disorder?	43
2.2.3.1 TD in non-clinical and healthy individuals	45
2.2.4 ‘Word salad’ or meaningful utterances?	47
2.2.5 ‘Hot’ and ‘cold’ thought disorder?	51
<i>2.3 Neurocognitive mechanisms and TD</i>	<i>59</i>
2.3.1 Executive ability	62
<i>2.4 Psychological mechanisms</i>	<i>67</i>
2.4.1 Semantic hyperpriming	67
2.4.2 Internal source monitoring	74
2.4.3 ‘Theory-of-Mind’	79
<i>2.5 Genes, heritability and thought disorder</i>	<i>85</i>

2.6 <i>The environment and thought disorder</i>	90
2.6.1 Trauma and adversity	91
2.6.2 Family communication	94
2.7 <i>Conclusions</i>	97
2.7.1 A transdiagnostic and very tentative cognitive-developmental model of TD	101
2.8 <i>References</i>	105
<b>Chapter 3. The affective reactivity of psychotic speech: The role of internal source monitoring in explaining increased thought disorder under emotional challenge</b>	<b>148</b>
3.1 <i>Abstract</i>	149
3.2 <i>Introduction</i>	150
3.2.1 Internal source monitoring (iSM)	150
3.2.2 Affect	151
3.3 <i>Materials and methods</i>	152
3.3.1 Participants	152
3.3.2 Materials	152
3.3.2.1 Psychotic symptoms	152
3.3.2.2 IQ	153
3.3.2.3 Interviews	153
3.3.2.4 TD	153
3.3.2.5 Affect	154
3.3.2.6 iSM	154
3.3.2.7 Immediate auditory recall	155
3.3.3 Procedure	155

3.3.4 Data analysis	158
3.4 Results	158
3.4.1 Demographic and clinical variables	158
3.4.2 Key study variables	158
3.4.3 iSM and affective reactivity in patients	161
3.5 Discussion	163
3.6 References	167
<b>Chapter 4. Inner speech and clarity of self-concept in thought disorder and auditory-verbal hallucinations</b>	<b>174</b>
4.1 Abstract	175
4.2 Introduction	176
4.2.1 TD as disruption of inner dialogue	177
4.2.2 Aims of the study	179
4.3 Methods	180
4.3.1 Participants	180
4.3.2 Measures	181
4.3.2.1 Psychotic symptoms	181
4.3.2.2 Quick Test	181
4.3.2.3 Interviews	182
4.3.2.4 TD	182
4.3.2.5 Self-concept clarity	182
4.3.2.6 Quality of Inner Speech	183
4.3.3 Procedure	184
4.3.4 Statistical analysis	184
4.4 Results	185

4.4.1 Demographic and clinical variables	185
4.4.2 Group differences on psychological measures	187
4.4.3 Clarity of self-concept and individual psychotic experiences	187
4.4.4 Inner speech and individual psychotic experiences	188
4.4.5 Dimensions of TD, inner speech, and clarity of self-concept	189
4.5 Discussion	191
4.5.1 Limitations	195
4.5.2 Implications for clinical practice	196
4.8 References	197
<b>Chapter 5. ‘No man is an island’: Testing the specific role of social isolation in thought disorder</b>	<b>213</b>
5.1 Abstract	214
5.2 Introduction	215
5.2.1 The relevance of formal thought disorder (FTD)	215
5.2.2 Psychological mechanisms in FTD	216
5.2.3 Social isolation and psychosis	217
5.2.4 Social isolation and specific symptoms	219
5.2.5 Aims of the present study	220
5.3 Methods	221
5.3.1 Participants and procedures	221
5.3.2 Assessment tools	225
5.3.2.1 Psychotic symptoms	225
5.3.2.2 IQ	226
5.3.2.3 Interviews	226
5.3.2.4 FTD	227

5.3.2.5 Social Isolation	227
5.3.3 Statistical analysis	228
5.4 Results	231
5.4.1 Demographics and clinical variables	231
5.4.2 FTD	231
5.4.3 Social Isolation	233
5.4.4 Social Isolation and symptoms	233
5.5 Discussion	236
5.5.1 Limitations	239
5.5.2 Clinical implications: From the lab to therapy	240
5.6 Bibliographic references	242
<b>Chapter 6. Communication deviance as an <i>envirotpe</i> for psychosis: A</b>	
<b>systematic review and an exploration of potential developmental pathways</b>	<b>261</b>
6.1 Abstract	261
6.2 Introduction	263
6.2.1 Communication deviance (CD)	264
6.3 Aims and objectives	275
6.4 Method	276
6.4.1 Search strategy	276
6.4.2 Exclusion criteria	276
6.5 Findings	277
6.5.1 Is CD in caregivers of patients with psychosis a replicable and specific phenomenon?	279
6.5.2 Is CD in caregivers associated with thought disorder (TD) in the offspring?	303

6.5.3 Is CD in caregivers associated with relapse in the offspring? _____	308
6.5.4 Is CD associated with other caregiver characteristics such as expressed emotion or psychopathology? _____	309
6.5.5 Can the association between CD in the caregiver and offspring psychopathology be explained by reverse-causality (the caregivers' reactions to their disturbed children)? _____	318
6.5.6 Can the association between CD in the caregiver and offspring psychopathology be explained by genetic factors? _____	322
6.6 <i>Conclusions from the review</i> _____	324
6.6.1 Limitations of CD research to date _____	324
6.6.2 The possible causal role of CD _____	325
6.6.3 Possible developmental pathways from CD to offspring psychopathology _____	327
6.6.3.1 Joint attention (JA) _____	331
6.6.3.2 Internalization of inner speech _____	334
6.6.3.3 Mind-mindedness _____	336
6.6.4 Conclusions about possible developmental pathways _____	338
6.7 <i>Bibliographic references</i> _____	342
<b>Chapter 7. Parental communication and psychosis: A meta-analysis</b> _____	<b>405</b>
7.1 <i>Abstract</i> _____	406
7.2 <i>Introduction</i> _____	407
7.3 <i>Methods</i> _____	411
7.3.1 Literature search strategy and eligibility criteria _____	411
7.3.2 Coding protocol and effect sizes (g) computation _____	417
7.4 <i>Results</i> _____	419

7.4.1 Pooled effect sizes and heterogeneity analysis	419
7.4.2 Publication bias	422
7.5 Discussion	424
7.6 References	431
<b>Chapter 8. Mapping early environment using communication deviance: A longitudinal study of maternal sensitivity towards 6-month-old children</b>	<b>459</b>
8.1 Introduction	460
8.1.1 Communication Deviance	460
8.1.2 Maternal sensitivity	463
8.1.2.1 Sensitivity to distress and to non-distress	465
8.1.3 Current study	465
8.2 Methods	466
8.2.1 Design	466
8.2.2 Participants	467
8.2.3 Measures and procedure	468
8.2.3.1 Communication Deviance (CD)	468
8.2.3.2 Maternal sensitivity at 29 weeks	472
8.2.3.3 Confounders	473
8.2.3 Statistical analysis	474
8.3 Results	478
8.4 Discussion	480
8.5 Bibliographic references	488
<b>Chapter 9. Conclusion</b>	<b>503</b>
9.1 Conclusions and future research	504
9.2 Bibliographic references	515





## List of tables

<b>Table 1</b> - Items from the scale for the assessment of thought, language and communication disorders _____	35
<b>Table 2</b> - Items of the different methodologies used to measure and quantify thought and communication disturbances _____	40
<b>Table 3</b> - Clinical and demographic variables _____	157
<b>Table 4</b> - Means and standard deviations for TD, iSM scores, DST and reported affect across group and interview _____	159
<b>Table 5</b> - Bivariate correlations between TD, iSM and affective scores for the clinical group _____	162
<b>Table 6</b> - Analysis of mediation, negative affect (x) on thought disorder (y) through discrimination index (m) for the clinical group _____	164
<b>Table 7</b> - Group data on clinical, demographic and psychological variables _____	186
<b>Table 8</b> - Bivariate correlations between TD, hallucinations, delusions, suspiciousness/persecution, negative symptoms, four dimensions of inner speech and clarity of self-concept (clinical group only) _____	189
<b>Table 9</b> - Partial correlations between TD factors, inner speech factors and clarity of self-concept after controlling for hallucinations, delusions, and suspiciousness/persecution (clinical group only) _____	192
<b>Table 10</b> - Clinical and demographic variables _____	223
<b>Table 11</b> - Frequencies and percentages of the TLC items across groups and interviews with group comparisons (with and without Bonferroni corrections) ____	230
<b>Table 12</b> - Means and standard errors for formal thought disorder (axis Y) scores in patients and comparisons across both neutral and salient interviews _____	233

<b>Table 13</b> - Partial correlations (Pearson's $r$ ) between psychotic symptoms, PANSS factors, formal thought disorder and social isolation after controlling for gender, age, years of education and verbal IQ (with and without Bonferroni correction)	235
<b>Table 14</b> - Two-stepped regressions with formal thought disorder ( $n= 80$ ) from both interviews as the outcome and hallucinations (P3), delusions (P1), suspiciousness (P6) and social isolation as predictors	237
<b>Table 15</b> - Communication deviance (CD) categories for Rorschach and TAT protocols (Jones, 1977; Singer & Wynne, 1966b; Wynne, Singer, Bartko, & Toohey, 1977)	267
<b>Table 16</b> - Some of the methodologies used to assess CD and related constructs	274
<b>Table 17</b> - Studies comparing parents of patients diagnosed with Schizophrenia with other parental groups.	293
<b>Table 18</b> - Characteristics of the studies included in the meta-analysis.	416
<b>Table 19</b> - Definitions and examples of the CD codes (Velligan, 1985)	470
<b>Table 20</b> - Frequencies and descriptives of the demographic variables in the study (unweighted)	477
<b>Table 21</b> - Means, standard deviations and ranges (weighted)	478
<b>Table 22</b> – Weighted bivariate regressions with CD as a predictor of overall maternal sensitivity and summary of the “bent-stick” regression model	479
<b>Table 23</b> - Weighted linear regression with CD as a predictor of overall maternal sensitivity after adjusting for maternal age and verbal IQ and index of multiple deprivation	481
<b>Table 24</b> - Weighted linear regression with CD as a predictor of maternal sensitivity to distress and non-distress after adjusting for maternal age and verbal IQ and index of multiple deprivation	484

## List of figures

<b>Figure 1</b> - Representation of a semantic network_____	69
<b>Figure 2</b> - Lexical decision task paradigm _____	71
<b>Figure 3</b> - A transdiagnostic and very tentative cognitive-developmental model of TD _____	102
<b>Figure 4</b> - Wynne's (1981) CD model_____	265
<b>Figure 5</b> - Flowchart of the studies included in the qualitative review_____	278
<b>Figure 6</b> - Schematic representation of two different developmental models of schizophrenia (Reichenberg et al., 2010)_____	328
<b>Figure 7</b> - Example of joint attention_____	335
<b>Figure 8</b> - Vygotsky's (1934) model of the development of verbal thought and higher cognition _____	336
<b>Figure 9</b> - A very hypothetical developmental pathway between CD and thought disorder_____	339
<b>Figure 10</b> - Flowchart of studies included in the meta-analysis_____	414
<b>Figure 11</b> – Hedges' g formula _____	417
<b>Figure 12</b> - Convention to calculate standard means difference from odds ratio ____	418
<b>Figure 13</b> - Forest plot _____	423
<b>Figure 14</b> - Regression model with lowess smooth, linear and “bent-stick” fit_____	481

## **List of appendices**

<b>Appendix 1</b> - Ethical approval, sponsorship letter and letters of access	523
<b>Appendix 2</b> - Consent form	543
<b>Appendix 3</b> - Participant information sheet (clinical)	544
<b>Appendix 4</b> - Participant information sheet (comparisons)	548
<b>Appendix 5</b> - Positive and Negative Syndrome Scale (PANSS)	552
<b>Appendix 6</b> - Psychosis Screening Questionnaire (PSQ)	565
<b>Appendix 7</b> - QUICK test (cards and words)	566
<b>Appendix 8</b> - Salient and non-salient interviews	569
<b>Appendix 9</b> - TLC scoring sheet	571
<b>Appendix 10</b> - Source monitoring (instructions, task, recognition sheet and example card)	572
<b>Appendix 11</b> - Positive and Negative Affect Scale (PANAS)	576
<b>Appendix 12</b> - Lubben's Social Network Scale (LSNS)	577
<b>Appendix 13</b> - Varieties of Inner Speech Questionnaire (VISQ)	580
<b>Appendix 14</b> - Self-concept Clarity Scale (SCCS)	582

## **Chapter 1. Introduction**

## 1.1 Introduction

Ever since Aaron Beck's early work on the treatment and psychological processes of delusions (Beck, 1952; Hole, Rush, & Beck, 1979), there has been a growing interest in the psychological study of psychotic experiences. This interest has led to the development of successful and useful psychological models of psychosis (Bentall, 2003; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001) that have identified not just atypical cognitive mechanisms and affective processes but also important social and environmental predictors of these experiences (Bentall & Fernyhough, 2008; Bentall et al., 2014).

Curiously, one experience, or cluster of experiences, that has been relatively neglected by cognitive scientists is formal thought disorder (TD). This relative neglect is strange given the long tradition of psychiatric research in this area (McKenna & Oh, 2005) with the first experimental studies dating back to the first half of the 20<sup>th</sup> century (Cameron, 1939). This neglect may have been motivated by the important conceptual and epistemological issues inherent to the study of TD (see Andreasen, 1982) as for example, the well-known debate centred around whether these experiences are linguistic, communicational or cognitive in nature (Chaika, 1982; Lanin-Kettering & Harrow, 1985; Rochester & Martin, 1979; Schwartz, 1982).

Despite the potential importance of these debates, the reality is that TD has clinical and real life significance given that it is a prevalent (Roche, Creed, MacMahon, Brennan, & Clarke, 2014) and enduring feature in patients diagnosed with psychotic-spectrum disorders (Bowie et al., 2005; Marengo & Harrow, 1987, 1997). Moreover, these experiences are associated with both poorer occupational (Racenstein, Penn, Harrow, & Schleser, 1999) and social functioning (Bowie, Gupta,

& Holshausen, 2011; Bowie & Harvey, 2008), poorer quality of life (Tan, Thomas, & Rossell, 2014) and higher relapse rates in psychotic patients (Wilcox, 1990).

Despite its relevance very little is known about the social and environmental predictors of TD and about the role of both cognitive and affective processes in these experiences. Such knowledge is important because it can inform the development of targeted therapeutic strategies to address TD (Beck, Rector, Stolar, & Grant, 2009). It is important to note that apart from a few studies (e.g. Satel & Sledge, 1989), there has been very little work on effective therapeutic strategies to address TD and by large the therapeutic techniques presented in manuals have relied heavily on therapist individual experience of what works (Beck et al., 2009; Hill & Temple, 2005; Stolar & Grant, 2011).

In the present dissertation, my aim was to address several gaps in knowledge. In **Chapter 2**, I start by reviewing some of the most well researched psychological mechanisms and affective processes as well as the more limited literature on environmental and social predictors of TD. Of note is the consistency of the evidence supporting the role of *internal source monitoring*, '*theory-of-mind*' and negative affect in TD and the limited literature on the social and environmental factors associated with these symptoms. On the latter issue, the historical literature on parental communication deviance (CD) stands out as the only reasonably well-replicated social predictor of TD. At the end of this section, we will present a diagram of a tentative cognitive-developmental model of TD that summarizes some of the research findings but also informs directions for future research.

In **Chapter 3**, I report on the investigation on the role of both *internal source monitoring* and negative affect in TD. This work stems directly from the review presented in **Chapter 2**, especially on the consistent findings supporting the crucial role of this cognitive mechanism and of negative affect in TD (all the studies reviewed on both processes yielded positive findings). My hypothesis was that the worsening of TD during emotional challenge (an emotionally-salient interview) in patients diagnosed with schizophrenia-spectrum disorders, would be predicted by negative affect (as it has been showed in previous studies); but also, and more importantly, that the relationship between negative affect and worsening of TD would be mediated by the temporary worsening of the patients ability to monitor self-generated cognitions (ability to differentiate between verbalized speech and inner speech i.e. internal source monitoring; Johnson, Hashtroudi, & Lindsay, 1993). In the discussion I suggest that instances of *disorganised* speech such as incoherence and derailment (believed to reflect a positive dimension of TD, see Andreasen & Grove, 1986; Cuesta & Peralta, 1999) could be understood as either the inadvertent verbalisation of inner speech or the omission of important segments of speech during emotionally challenging circumstances (where the “jumbled up” quality of the TD could be conceptualized as the condensed and agrammatical nature of inner speech).

In **Chapter 4**, I report the findings of an exploratory study where the role of inner speech and self-concept in TD are explored. My hypothesis was that different dimensions of TD would be differentially associated with the two variables. Of interest is the fact that the analyses revealed that poverty of speech dimension of TD was strongly associated with less reported dialogical, evaluative, and other people in inner speech (perhaps suggesting that poverty of speech may be best described as



poverty of verbal thought). In contrast, poor clarity of self-concept was significantly associated with disorganised forms of TD.

In **Chapter 5**, I report the findings of another set of analyses where the specific role of social isolation in TD was tested. The analyses were motivated by the hypothesis that lack of social interaction and communication are likely to have crucial implications for this cluster of symptoms (social interaction and communication are crucial for the development of social skills and are likely to have a detrimental impact on the patients' communication, linguistic abilities and more generally speaking cognition). Although social isolation, as a risk factor, has been often associated with psychosis and specific psychotic experiences (e.g. hallucinations and delusions) a specific association with TD has never been properly tested. The analyses indicated that social isolation was robustly associated with TD, and more importantly, that the association remained significant even when comorbid psychotic symptoms (hallucinations and delusions) were controlled for statistically.

In **Chapter 6**, I present a systematic review of the field of communication deviance (CD). The review covers not just case-control studies but also adoption studies that have supported the association between parental CD and offspring's TD, as initially suggested by Wynne and Singer (Singer & Wynne, 1965a, 1965b; Wynne & Singer, 1963a, 1963b). I try to address some of the questions and methodological issues that have been raised by authors in the field (e.g. its relationship with other family variables such as expressed emotion, its prevalence across parents of offspring with different diagnosis, etc.) and argue that CD is an important and specific environmental risk factor for TD. In trying to move the field forward, I advance some plausible developmental pathways and mechanisms that could potentially explain the relationship between parental CD and offspring's TD and cognitive problems.

In **Chapter 7**, I present a meta-analysis of the studies published between 1959 and 2012 that have tested CD in parents of psychotic offspring and controls. Of note is the significant and robust pooled effect-size found, and perhaps more importantly, its stability when I re-analysed the data taking into consideration the different methodological features (e.g. year of publication, study design, age of the offspring, CD methodology, inter-rater reliability, verbosity, etc.). The sub-analysis of the effect-sizes for mothers and fathers revealed that CD was significantly more prevalent in the former group. Unfortunately, there was not enough data to carry out a meta-analysis looking specifically at the association between parental CD and offspring's TD.

In **Chapter 8**, I report the findings of a prospective cohort study where I tested the association between CD measured in the speech of primiparous mothers at 32 weeks into their pregnancy and maternal sensitivity measured at 29 weeks while these mothers played with their infants. The analyses revealed robust and significant associations between CD and maternal sensitivity in the context of infant's distress during a lab-based play protocol. In the discussion of the findings, I advance a possible developmental pathway linking CD and maternal sensitivity to specific cognitive mechanisms in the offspring that are relevant to TD.

Finally, in **Chapter 9**, I present the conclusions and try to integrate both social predictors and psychological mechanisms of TD with the aim to promote potential avenues for future research.

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## **Chapter 2. The psychology of thought disorder: A narrative review**

This paper is currently in preparation as de Sousa, P., Sellwood, W., & Bentall, R. P.  
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“Thought is not merely expressed in words; it comes into existence through them. Every thought tends to connect something with something else, to establish a relationship between things. Every thought moves, grows and develops, fulfils a function, solves a problem.” (Vygotsky, 1934, p. 125).

## 2.1 Introduction

Formal thought disorder (TD) is a term that is widely used in the mental health context in Anglophone countries. The term refers to a rather heterogeneous range of communicational, linguistic and cognitive atypicalities (Andreasen & Grove, 1986; Cuesta & Peralta, 1999) that render the communication and speech of some psychotic patients difficult to follow and *apparently* unintelligible (Andreasen, 1982).

The descriptor “formal” (that precedes “thought disorder”) is used to differentiate these atypicalities from “disorders of thought content” such as delusional beliefs (Barrera & Berrios, 2001). Implicit to this nosological subtlety is the idea that TD refers to disturbances in the “flow” of thought and speech or to the “formal” qualities of the thinking processes rather than the content of thought (e.g. acceleration, retardation, interruption or changes to the flow of thought, Sims, 1988).

The study and characterization of TD has long interested psychopathologists and clinicians. Classical psychopathologists such as Bleuler (1911) and Kraepelin (1913) have provided early detailed clinical descriptions of TD (e.g. Kraepelin described *akataphasia* as a dissolution of the logical ordering of the train of thought that was believed to be a symptom of *dementia praecox*) and not long after, these accounts were followed by early experimental work (e.g. Cameron, 1938).

Possibly due to the lasting influence of Eugen Bleuler in the world of Anglophone psychiatry, for a long time TD and *loosening of associations* (often used interchangeably) were believed to be a primary and core symptom of schizophrenia (Andreasen, 1982; Barrera & Berrios, 2001; Peralta & Cuesta, 2011) motivating much of the early interest in TD (e.g. the early word-association tests or the overinclusive thinking-hypothesis of TD, *see* Schwartz, 1982).

Over the years an impressive number of models and theories have been proposed to explain TD (Chapman & Chapman, 1973; Goldberg & Weinberger, 2000; Harrow & Quinlan, 1985; Johnston & Holzman, 1979; McKenna & Oh, 2005; Schwartz, 1982) and many more have not survived the test of time (Reed, 1970). This effort produced several now outdated models and spurious and redundant terms (Rule, 2005) leading some authors to question the utility of the term “TD” (Andreasen, 1982). However, and despite conceptual and epistemological obstacles (*see* Szasz, 1993), research on TD is important for several reasons:

- (1) TD is highly prevalent in psychotic patients with some estimates reaching 65%, 72.7% or even 95% (Andreasen & Grove, 1986; Andreasen, 1979a; Cuesta & Peralta, 2011; Roche, Creed, MacMahon, Brennan, & Clarke, 2014);
- (2) TD has been found to be highly predictive of future relapse in psychotic patients (Wilcox, 1990), a picture that is further complicated by the relative lack of evidence-based therapeutic strategies to address it (Beck, Rector, Stolar, & Grant, 2009; Stolar & Grant, 2011);
- (3) TD is associated with poorer occupational functioning (Racenstein, Penn, Harrow, & Schleser, 1999), poorer social functioning (Bowie, Gupta, &

Holshausen, 2011; Bowie & Harvey, 2008; Smith et al., 1999), poorer quality of life (Tan, Thomas, & Rossell, 2014) and this state of affairs is further complicated by its persistent course (Bowie et al., 2005; Docherty, Cohen, Nienow, Dinzeo, & Dangelmaier, 2003; Harrow & Marengo, 1986; Jampala, Taylor, & Abrams, 1989; Marengo & Harrow, 1987, 1997; Wilcox, Winokur, & Tsuang, 2012; Winokur, Scharfetter, & Angst, 1985); (4) TD seems to be an early predictor of later conversion into psychosis in at-risk populations (Bearden, Wu, Caplan, & Cannon, 2011; Cannon et al., 2008; Gooding et al., 2012; Gooding, Ott, Roberts, & Erlenmeyer-Kimling, 2012; Ott, Roberts, Rock, Allen, & Erlenmeyer-Kimling, 2002; Ruhrmann et al., 2010) providing clinicians with a potential window of opportunity for preventive work.

Finally, research on the psychological mechanisms and social predictors of TD is likely to inform the much-needed development of therapeutic techniques to address this symptom. It is worth mentioning that apart from a couple of studies focused on the use of video-taped feedback (Satel & Sledge, 1989) and reinforcement (Meichenbaum, 1969) very little has been done to develop specific therapies for TD.

## **2.2 The nature of thought disorder**

### **2.2.1 What is so disordered about thought disorder?**

In 1974, Andreasen, Tsuang and Canter asked a group of psychiatrists and experienced mental health professionals to blindly rate six speech samples for the presence of TD. Four of these transcripts were proverb interpretations made by

patients diagnosed with either schizophrenia or bipolar affective disorder (BPAD) and the other two were excerpts from a poem by Marvin Bell (*The perfection of dentistry*, 1969) and a section of James Joyce's cryptic novel *Finnegans Wake*<sup>1</sup> (1939).

Curiously, but perhaps unsurprisingly, 95% and 52% of the professionals identified the presence of TD in James Joyce's excerpt and in Marvin Bell's poem, respectively.

The primary goal of the study was to demonstrate that TD had poor diagnostic value, alluding to the long held belief that creativity and TD are closely related (Reed, 1970; Weeks & James, 1995). However, what the results from the study really called into question was the meaning of the TD. How can something that is portrayed as meaningful literary art in one context be considered a symptom of psychopathology in a different context?

So what is TD? TD is an umbrella term for a range of speech, communicational, and cognitive disturbances frequently observed in psychotic patients (Andreasen, 1982). The term is quite encompassing, ranging from *neologisms* i.e. words coined by the individual that do not have a socially accepted meaning; to *incoherence* i.e. speech that is essentially incomprehensible to the listener. The cohesive feature at the core of this construct is that these disturbances make speech hard to follow and often unintelligible to the listener. In the late seventies to mid-eighties, Andreasen reviewed the terminology used in the mental health field and developed what is still the most used and consensual methodology for assessing TD namely, the *scale for the assessment of thought, language and communication disorders* (TLC, Andreasen & Grove, 1986; Andreasen, 1979a, 1979b, 1986). Table 1 lists the 18 items of the scale with their respective definition and examples.

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<sup>1</sup> *Finnegans Wake* and *The Perfection of dentistry* are excellent examples of *stream-of-consciousness* writing style. This is a narrative technique where the thoughts, images and feelings of the different characters are expressed as inner experience, not always respecting conventional logic or grammatical structure.

TLC item	Definition	Example
<b>Poverty of speech</b>	A pattern of speech marked by a marked reduction in the amount of spontaneous speech. Replies to questions are often very brief and lack detail.	"(Do you think there's a lot of corruption in government?) Yeah, seem to be." (Andreasen, 1986, p. 474).
<b>Poverty of content of speech</b>	Pattern of speech that is vague, too general in content, and that conveys very little meaning.	"My goal is to do things that I want to do in my life so that I can accomplish them and know that I did them while I was alive and having goals."(Beck et al., 2009, p. 291).
<b>Pressure of speech</b>	Speech that is atypically fast. Speaker makes very few pauses and is very difficult to interrupt.	“(Are you a collector?) Yes. I do...the collection comes down to this...If someone says to me I have a rhyme for your CB broadcast...my first answer would be...did you? Or the question would be...did you do it yourself? Now, I am interested if he or she did it themselves...it doesn't matter what it sounds like, what is of interest to me is what it sounds like, but it is more important that they did it themselves. If it is something say Wordsworth...then I am sorry. I am not interested. I am only interested in the immediate...what you or your experience. If I want Wordsworth then I suppose I would go and get him and explain it to myself one way or another, but I don't want that, but I do want this one. That is the difference....I collect that...I don't collect Wordsworth, but I do collect the aspiring or the clever. I will take anything – virtually the most prosaic thing, and I can kick it into a story, for instance, verses really to me are little stories, it just so happens that I put them into verse, maybe one verse. If so then these are the most difficult, because you have to get a beginning, a middle, and an end, all in one verse – if you have got more – I take any subject like that.”(in rapid tempo) (Weeks & James, 1995, pp. 207–208).
<b>Distractible speech</b>	The topic of speech is abruptly interrupted and swapped by a topic related to stimuli in the immediate environment.	"Then I left San Francisco and moved to . . . Where did you get that tie? It looks like it's a left over from the fifties. I like the warm weather in San Diego. Is that a conch shell on your desk? Have you ever gone scuba-diving?" (Andreasen, 1986, p. 476).
<b>Tangentiality</b>	The speaker replies to a question in a way that is only vaguely related to the topic.	“(Strike while the iron is hot.) It could mean (pause) Hercules! (Could you say more?) I saw the movie Hercules. (Yes...) and it means don't iron over your hands and don't strike anybody before you cast the first stone.” (Marengo, Harrow, Lanin-Kettering, & Wilson, 1986, p. 498).
<b>Derailment</b>	An unpredictable pattern of speech in which speaker abruptly wanders off onto different and unrelated topics.	“(How are you?) To relate to people about new-found...talk about statistical ideology. Err...I find that it's like starting in respect of ideology, ideals change and ideals present ideology and...new entertainments...new, new attainments. And the more one talks about like, ideal totalitarianism or hotelatarianism, it's like you want new ideas to be formulated, so that everyone can benefit in mankind, so we can all live in our ideal heaven. Presumably, that's what we still want, and with these ideas it can be brought about, I find the...it's like a rose garden.”(Laws, Kondel, & McKenna, 1999, p. 105) .
<b>Incoherence</b>	A pattern of speech that is totally incoherent and grossly unintelligible.	“Yes, they add up and kind of like a solution. It's say, it's an equine or equinox, like fungi. Something in the brain tells you it's a high number. Bacteriology, a numerate number, it's a particle, therefore it contains solution is to answer the right question. A fork is a solution, an aqueous solution. Fork in a kettle, something bottle, do hairs bristle on a comb or fungi? It could be naval or positive solution ratified like a kettle, if kettle is the right answer. It could be 5 <sup>th</sup> or 7 <sup>th</sup> one, right? Brown aqueous solution inside the kettle.” (Laws et al., 1999, p.105).

<b>Illogicality</b>	A pattern of speech marked by inferences that are illogical.	“Parents are the people that raise you. Anything that raises you can be a parent. Parents can be anything, material, vegetable, or mineral, that has taught you something. Parents would be the world of things that are alive, that are there. Rocks, a person can look at a rock and learn something from it, so that would be a parent.” (Andreasen, 1986, p. 478).
<b>Clanging</b>	A pattern of speech in which words are associated by their phonological resemblance rather than their meaning.	"I'm not trying to make noise. I'm trying to make sense. If you can make sense out of nonsense, well, have fun." "I'm trying to make sense out of sense. I'm not making sense [cents] anymore. I have to make dollars."(Andreasen, 1986a; p. 478).
<b>Neologism</b>	Newly created word that does not have a socially accepted meaning and therefore is unknown to the listener.	“I felt a little <u>dizziwhelmed</u> .”(Beck et al., 2009; p. 289).
<b>Word approximations</b>	Words that are used in an unconventional and idiosyncratic way.	“I have since resolved to actually <u>Sherlock Holmes</u> a manuscript...”(Weeks & James, 1995, p. 202).
<b>Circumstantiality</b>	A pattern of speech that is delayed getting to the point and that is marked by excessive and irrelevant details.	“(Tell me about your work. What do you like about it the most, and why?) Oh, it's easy work for the pay. I guess that's what I like about it the best. It has good benefits. Uh, my hours are pretty good, except when I have to work 8-hour days. Sometimes I get stuck with the 10 to 7 shift. That's kind of a drag. Urn, it's mostly older people that come into the grocery store, since it's up in the Polish neighbourhood, I guess, of Iowa City. I think they come there mostly for somebody to talk to. They don't have anything else to do, most of them are retired. Uh, I like to sew. I went and got a new sewing machine this week, and I plan on making a dress for Noelle for Christmas. I like to swim, I haven't had the chance since I was pregnant and I haven't gone back yet I like little kids. I hope I'll have some more, but I don't know whether I'll be able to afford it Um, let me think. I voted today”(Andreasen & Grove, 1986, pp. 351–352).
<b>Loss of goal</b>	Pattern of speech in which thoughts don't follow into a conclusion and ideas are left pending without closure.	“I want to talk about going back to school. I went to school when I was young. I have a younger brother. He lives in Oregon.”(Beck et al., 2009, p. 289).
<b>Perseveration</b>	One word and idea are persistently repeated in an irrelevant and decontextualized way.	"(Tell me what you are like, what kind of person you are.) I'm from Marshalltown, Iowa. That's 60 miles northwest, northeast of Des Moines, Iowa. And I'm married at the present time. I'm 36 years old. My wife is 35. She lives in Garwin, Iowa. That's 15 miles southeast of Marshalltown, Iowa. I'm getting a divorce at the present time. And I am at presently in a mental institution in Iowa City, Iowa, which is a hundred miles southeast of Marshalltown, Iowa."(Andreasen, 1986, p. 479).
<b>Echolalia</b>	Speaker mechanically echoes the last words or sentence of the interviewer without any apparent communicational intent.	“(How long have you lived there?)...have you lived there.”(Beck et al., 2009, p. 291).

<b>Blocking</b>	The abrupt and complete interruption of the flow of speech that can last for seconds or minutes. After the interruption, speaker is unable to return to original idea.	"I took a walk in the park...(long pause)...someone was walking a dog."(Beck et al., 2009, p. 291).
<b>Stilted speech</b>	A pattern of speech that is stilted and marked by excessive formality for its context.	"My errors may have compounded my naiveté lack of worldly wise. To add fear to fearfulness is cruelty." (Weeks & James, 1995, p. 202).
<b>Self-reference</b>	The speaker repeatedly refers topic back to himself whilst discussing reasonably neutral issues.	"(What time is it?) Seven o'clock. That's my problem. I never know what time it is. Maybe I should try to keep better track of the time."(Andreasen, 1986, p. 480).

**Table 1** - Items from the scale for the assessment of thought, language and communication disorders.



Looking at items such as *stilted speech* and *circumstantiality* it becomes evident that the judgment on whether someone is thought-disordered or not is often dependent on the context. For example, what a mental health professional may consider *stilted speech* in a psychotic patient may be a perfectly appropriate way of addressing the UK House of Commons or US Senate. In the same way, *neologisms* are constantly being created in ordinary language. Shakespeare, for example, coined or at least recorded for the first time 2,035 words, including: “*abstemious, antipathy, critical, frugal, dwindle, extract, horrid, vast, hereditary, excellent, eventful, barefaced, assassination, lonely, leapfrog, indistinguishable, well-read, zany*, and countless others (including *countless*)” (Bryson, 2007).

This point is extremely important given the recent recognition that psychotic experiences are much more widely distributed in the general population than was once believed (see 2.3.1. below), recent research showing a (likely at least partially genetic) association between psychosis and creativity (Kyaga et al., 2013; Power et al., 2015) and the increasingly recognized ethical imperative to ‘depathologise’ the language of mental health (Kinderman, Read, Moncrieff, & Bentall, 2013). With this in mind, the authors opted to keep the term TD only for its clinical and historical value remaining highly conscious of its limitations (Rochester & Martin, 1979; Szasz, 1993).

### **2.2.2 TD as a heterogeneous construct**

Over the years a variety of theories and models have been proposed to explain the mechanisms involved in TD. These have ranged from a deficit in abstract thinking (Goldstein, 1944), a deficit of syllogistic reasoning (Von Domarus, 1944), a deficit in selective attention (Chapman & McGhie, 1962), excessive attention to stimuli in the immediate environment (Salzinger, Portnoy, Pisoni, & Feldman, 1970), excessive yielding to

normal bias in face of ambiguous stimuli (Chapman, Chapman, & Miller, 1964), a thinking pattern marked by overinclusiveness (Cameron, 1939), the consequence of a loosening of personal constructs (Bannister, 1960) or of semantic generalization due to anxiety (Mednick, 1958), a style of communication learnt from the family (Singer & Wynne, 1965a, 1965b; Wynne & Singer, 1963a, 1963b), specific difficulties establishing and maintaining cognitive set (Rodnick & Shakow, 1940), a lack of cohesion in discourse (Rochester & Martin, 1979), a speech disorder (Chaika, 1982), a genetic vulnerability with expression at the level of semantics (Levy et al., 2010), a deficit in discourse planning (Hoffman, 1986), the consequence of a dysexecutive syndrome (McGrath, 1991) to a failure editing-out speech (Cohen, 1978) or source-monitoring self-generated cognitions (Harvey, 1985).

Most of these theories are in fact compatible and some of them have been incorporated into more sophisticated models which we will discuss below. However, the terminology and consequently the methodologies used to assess TD have remained confusing. For example, some scoring schemes or rating scales were developed for speech samples acquired through Rorschach protocols or subtests from Wechsler Adult Intelligence Scale (e.g. similarities) (see Thought Disorder Index, Solovay et al., 1986) or the Thematic Apperception Test (see Bizarre-idiosyncratic thinking scale, Harrow & Quinlan, 1985; or Thought Language Index, Liddle et al., 2002) whereas other scoring schemes were developed for more conversational speech samples (see Clinical Language Disorder Rating Scale, Chen, Lam, Kan, & Chan, 1966; or Communication Disturbances Index, Docherty, 1996b).

Table 2 details the items of 5 different methodologies frequently used to assess TD and related constructs. In some cases, the items refer to inferred disturbed thought processes (e.g. *autistic logic* in Thought Disorder Index or *non-logical reasoning* in Thought Language Index) whereas in other cases they target linguistic (e.g. *excessive syntactic constraints* in Clinical Language Disorder Rating Scale) and communicational disturbances (e.g. *vague*

*references* in Communication Disturbances Index). At the core of these differences is the debate of whether TD is a communicational, a linguistic or a cognitive disturbance (Chaika, 1982; Lanin-Kettering & Harrow, 1985; Rochester & Martin, 1979).

In the perspective of the authors, this debate is not particularly relevant. If it is true that some features appear to be linguistic (Covington et al., 2005) it is also clear that some disturbances can only be explained at a more conceptual level e.g. *illogicality* (Lanin-Kettering & Harrow, 1985). Also, TD seems to be strongly associated with *incongruent* non-verbal behaviour, bizarre ideas (Harrow et al., 2003) and symptom-dimension *cognitive disorganisation*, which encompasses non-linguistic experiences such as *inappropriate affect* or *bizarre behaviour* (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; Liddle, 1987; Reininghaus, Priebe, & Bentall, 2012; van Os & Kapur, 2009). Other studies have found significant associations between TD and disturbances at the level of emotional processing (Kerns & Becker, 2008), suggesting that a purely linguistic model may not be sufficient to explain the complexities of the construct. And if it is true that linguistic studies have reported that the speech of patients diagnosed with schizophrenia tends to be less syntactically complex (Fraser, King, Thomas, & Kendell, 1986; Morice & Ingram, 1982; Sanders, Adams, & Tager-Flusberg, 1995) and more grammatically deviant (Hoffman & Sledge, 1988) it is also true that these linguistic characteristics seem to be associated with negative symptoms and duration of the illness rather than being specific to TD (King, Fraser, Thomas, & Kendell, 1990; Morice & Ingram, 1983; Thomas, King, & Fraser, 1987; Thomas, King, Fraser, & Kendell, 1990).

Thought disorder index (TDI; Johnston & Holzman, 1979)	Thought language index (TLI; Liddle et al., 2002)	Bizarre-idiosyncratic thinking scale (BIT; Harrow & Quinlan, 1985)	Clinical language disorder rating scale (CLANG; Chen, Lam, Kan, & Chan, 1996)	Communication disturbances index (CDI; Docherty, 1996)
1. Inappropriate distance <b>a. Loss or increase of distance</b> <b>b. Excessive qualification</b> <b>c. Concreteness</b> <b>d. Over specificity</b> <b>e. Syncretistic response</b> 2. Flippant response 3. Vagueness 4. Peculiar verbalisations and responses <b>a. Peculiar expression</b> <b>b. Stilted, inappropriate expression</b> <b>c. Idiosyncratic word usage</b> 5. Word-finding difficulty 6. Clangs 7. Perseveration 8. Incongruous combinations <b>a. Composite response</b> <b>b. Arbitrary form-colour response</b> <b>c. Inappropriate activity response</b> <b>d. External-internal response</b> 9. Relationship verbalisation 10. Idiosyncratic symbolism <b>a. Colour symbolism</b> <b>b. Image symbolism</b> 11. Queer responses <b>a. Queer expressions</b> <b>b. Queer imagery</b> <b>c. Queer word usage</b> 12. Confusion 13. Looseness 14. Fabulized combinations, impossible or bizarre	<b>Poverty of speech</b> <b>Weakening of goal</b> <b>Perseveration of ideas</b> <b>Looseness</b> <b>Peculiar use of words</b> <b>Peculiar sentences</b> <b>Non-logical reasoning (peculiar logic)</b> <b>Distractibility</b>	<b>I. Linguistic form and structure</b> 1. Peculiar word form or use 2. Lack of shared communication <b>II. Content of the statement: the ideas expressed</b> 3. Coherent but odd ideas 4. Deviant with respect to social convention 5. Peculiar reasoning or logic 6. Confused ideas <b>III. Intermixing tendencies</b> 7. The over-elaborated response 8. Intermingled response <b>IV. The relation between question and response</b> 9. Attention to limited part of the stimulus 10. The lack of relation between the subject's statement and the question asked <b>V. Behaviour</b>	<b>Excess phonetic association</b> <b>Abnormal syntax</b> <b>Excess syntactic constraints</b> <b>Lack of semantic association</b> <b>Referential failures</b> <b>Discourse failure</b> <b>Excess details</b> <b>Lack of details</b> <b>Aprosodic speech</b> <b>Abnormal prosody</b> <b>Pragmatics disorder</b> <b>Dysfluency</b> <b>Dysarthria</b> <b>Poverty of speech</b> <b>Pressure of speech</b> <b>Neologisms</b> <b>Paraphasic error</b>	<b>Vague references</b> <b>Confused references</b> <b>Missing information references</b> <b>Ambiguous word meanings</b> <b>Wrong word references</b> <b>Structural unclarities</b>

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15. Playful confabulation
16. Fragmentation
17. Fluidity
18. Absurd responses
19. Confabulations
<b>a. Details in one area generalized to larger area</b>
<b>b. Extreme elaboration</b>
20. Autistic logic
21. Contamination
22. Incoherence
23. Neologisms

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**Table 2** - Items of the different methodologies used to measure and quantify thought and communication disturbances.

Clearly, one reason for the persistence of this debate is the fact that TD is a multi-dimensional construct (Cuesta & Peralta, 1999) which suggests that a single-mechanism model is not sufficient to account for its diversity and complexity, as McKenna and Oh (2005) suggest:

“(...) thought disorder is itself not a unitary clinical phenomenon. If any attempt to reduce different elements of thought disorder to a single phenomenon is doomed to failure, then there is no reason to expect a single cognitive abnormality to reproduce it in all respects – in some ways this is the last thing that is wanted.” (p. 181).

Several studies have been carried out to test the different dimensions of TD. In many of these studies, authors have identified a negative dimension generally associated with underproductive speech (Andreasen & Grove, 1986a; Cuesta & Peralta, 1999; Harvey, Earle-Boyer, & Wielgus, 1984; Liddle et al., 2002; Nagels et al., 2013; Peralta, Cuesta, & de Leon, 1992) and a positive dimension associated with disorganisation of the flow of thought (Andreasen & Grove, 1986; Andreou et al., 2008; Bazin, Lefrere, Passerieux, Sarfati, & Hardy-Bayle, 2002; Berenbaum, Oltmanns, & Gottesman, 1985; Cuesta & Peralta, 1999; Harvey et al., 1992, 1984; Liddle et al., 2002; Nagels et al., 2013; Taylor, Reed, & Berenbaum, 1994).

These statistical dimensions have been replicated irrespective of the scale used and are consistent with a more conceptual analysis of the different categories of TD (Berenbaum & Barch, 1995). However, the reported factor solutions have generally varied from 2 (Harvey et al., 1984; Taylor et al., 1994) and 3 (Andreasen & Grove, 1986; Andreou et al., 2008; Berenbaum et al., 1985; Harvey et al., 1992; Liddle et al.,

2002; Nagels et al., 2013) to 5 or more (Bazin et al., 2002; Peralta et al., 1992; Solovay et al., 1986).

In probably the most comprehensive study to date, Cuesta and Peralta (1999) tested several dimensional models of TD using the TLC (Andreasen, 1986) and data from 253 participants diagnosed with schizophrenia-spectrum disorders (DSM-III-R, APA, 1987). The model that provided the best statistical fit incorporated 6 dimensions:

1. Negative dimension (poverty of speech, poverty of content of speech, and perseveration);
2. Idiosyncratic dimension (word approximations and stilted speech);
3. Semantic dimension (clanging and neologism);
4. Attentional dimension (distractible speech and blocking);
5. Referential dimension (echolalia and self-reference);
6. Disorganisation dimension (pressure of speech, tangentiality, derailment, incoherence, illogicality, circumstantiality, and loss of goal).

The items that were more prevalent and that explained more variance were the ones associated with the negative- and disorganisation-dimensions, suggesting that these may be the core dimensions of TD. TLC items such as *clanging*, *neologisms*, *word approximations*, *echolalia*, *blocking*, *stilted speech*, and *self-reference*, that did not load on either the negative- or the disorganisation-dimensions, had a prevalence of only 3-19%.

### **2.2.3 What is so “schizophrenic” about thought disorder?**

As mentioned earlier, for a long time TD was believed to be a core symptom of schizophrenia (Andreasen, 1982; Peralta & Cuesta, 2011; Barrera & Berrios, 2001) and some authors went a step further to suggest that the different symptoms of TD could potentially differentiate between different psychotic and affective disorders (Cuesta & Peralta, 1993; Holzman, Shenton, & Solovay, 1986; Shenton, Solovay, & Holzman, 1987; Solovay, Shenton, & Holzman, 1987). However, several studies have reported no significant differences in TD between participants diagnosed with schizophrenia and BPAD (Andreasen, 1979; Andreasen & Grove, 1986; Küfferle, Lenz, & Schanda, 1985; Oltmanns, et al., 1985; Simpson & Davis, 1985). For example, Harrow and colleagues (1982) reported that BPAD participants were as thought-disordered as participants diagnosed with schizophrenia upon admission to a mental health ward. In another study, Harvey (1983) tested participants diagnosed with BPAD and schizophrenia on TD (TLC, Andreasen, 1986), cohesive ties in discourse and deictic linguistic references (Rochester & Martin, 1979). The author reported no significant differences between thought-disordered participants across diagnostic groups. In fact, the only differences were between thought and non-thought disordered participants irrespective of their diagnostic group. Marengo and Harrow (1985) also reported a comparable amount of TD in participants diagnosed with both BPAD and schizophrenia. Interestingly, they also reported significant amounts of TD (bizarre-idiosyncratic thinking; Harrow & Quinlan, 1985) in healthy controls and non-psychotic participants<sup>2</sup> (which included diagnoses ranging from depression to OCD) suggesting that TD is not exclusively associated with psychosis.

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<sup>2</sup> 30% of the healthy comparisons and 41% of non-psychotic controls had either ‘signs of abnormal thinking’ or severe TD.



Several studies have also tested the presence of TD in participants diagnosed with autistic-spectrum disorders (Dykens, Volkmar, & Glick, 1991; Gaag, et al., 2005; Rumsey, Andreasen, & Rapoport, 1986). This may be of special relevance given that autistic-spectrum individuals have significant difficulties in their socio-cognitive and ‘theory-of-mind’ abilities (Baron-Cohen, 1997) and such difficulties have been suggested to be of special relevance to TD (Frith, 1992; Hardy-Baylé, Sarfati, & Passerieux, 2003).

In one of these studies, Rumsey, Andreasen, and Rapoport (1986) reported significantly higher TLC rates of *poverty of speech*, *poverty of content of speech* and *perseveration* in autistic participants in comparison with healthy comparisons. These results have been partially replicated by Dykens, Volkmar, and Glick (1991) who also reported that autistic-spectrum participants had higher rates of *poverty of speech* compared to participants diagnosed with schizophrenia and with differences on *loss of goal* and *derailment* not achieving statistical significance.

Finally, Gaag and colleagues (2005) reported that autistic children displayed significantly more TD (*illogicality* and *loose associations*) on the Kiddie-Formal Thought Disorder Scale (K-FTD; Caplan, et al., 1989) than healthy comparisons and children diagnosed with ADHD and anxiety disorders. These results have since been replicated (Solomon, et al., 2008).

In the line with a transdiagnostic view of TD, other researchers have tested its prevalence across a range of different diagnostic groups. For example, one study reported evidence of significant levels of TD in participants diagnosed with OCD (Lee, Kim, & Kwon, 2005). In this study, OCD participants with *autogenous*

*obsessions*<sup>3</sup> had levels of TD on the Rorschach that were comparable to the schizophrenia group. Edell (1987) reported clinical levels of TD (TDI; Johnston & Holzman, 1979) in participants diagnosed with borderline personality disorder (BPD). Again, the differences between this group of participants and groups of participants diagnosed with schizophrenia and schizotypal personality disorder did not reach statistical significance. Other studies have reported significant prevalence of TD in schizotypal personality disorder (Caplan, et al., 1990; Handest & Parnas, 2005). Caplan and colleagues (2001) reported that children diagnosed with ADHD displayed significant amounts of *illogical thinking* (K-FTD; Caplan, et al., 1989) when compared to healthy comparisons. Whereas Smith, Hillard and Roll (1991) reported a sizable and significant amount of TD on the Rorschach in participants diagnosed with eating disorders. Similar findings have also been reported for depression (Ianzito, Cadoret, & Pugh, 1974) and organic conditions such as *delirium* (Cutting, 1987) and epilepsy (Caplan, et al., 1997; Caplan, et al., 1992; Caplan, et al., 2006).

#### ***2.2.3.1 TD in non-clinical and healthy individuals***

Over recent years, epidemiological (e.g. Van Os, Hanssen, Bijl, & Ravelli, 2000) and psychological studies (Claridge, 1990) have offered support to the idea that psychotic symptoms occur on a continuum with healthy functioning, with no real discrete separation between clinical and general populations. Although some researchers continue to insist that the continuum model remains unproven (David, 2010) or that discrete diagnoses such as ‘schizophrenia’ are useful for pragmatic purposes (Lawrie, Hall, McIntosh, Owens, & Johnstone, 2010), the balance of research evidence favours at least a phenomenological continuum (that subclinical varieties of psychotic

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<sup>3</sup> Autogenous obsessions are ego-dystonic thoughts, impulses or images of aversive content (e.g. sexual, aggressive content, etc.) that erupt to consciousness with a recognizable trigger or evoking stimuli.

behaviour and experience can be identified in healthy individuals) although the existence of a structural continuum remains more contested, with some evidence pointing to a ‘taxon’ of individuals who are especially vulnerable to these kinds of experiences (Van Os, et al., 2009). Taxometric studies have so far failed to resolve this issue, partially because of unresolved debates about appropriate methods (Lenzenwenger, 2010; Rawlings, Williams, Haslam, & Claridge, 2008).

Several authors have suggested that TD may be dimensional (Liddle, et al., 2002; Reed, 1970), but this issue has been less addressed than for other types of psychotic experience. For example, Andreasen and Grove (1986) had reported quite a significant prevalence of *derailment* (32%) and *loss of goal* (18%) in healthy participants (n= 94). In this study, participants had been recruited through an advert in a local newspaper and had all been screened for psychotic and affective disorders (Research Diagnostic Criteria; Spitzer, Endicott, & Williams, 1979). This finding is by no means unique since other authors have also reported similar results. For example, Liddle and colleagues (2002) reported the presence of disorganised and impoverished forms of TD in their sample of healthy controls. What was perhaps more striking was that these levels of TD were by no means negligible. In the discussion of the study, the authors wrote:

“(...) there might be a continuum of severity of disorganised thought in the human population. The occurrence of such a continuum would have important implications for the nature of psychotic thought disorder, suggesting that this disorder might arise from one or more causal factors that exert an influence widely in the population.” (p. 329).

Weeks and James (1995) reported a high prevalence of TD in a sample of self-defined non-clinical *eccentrics*<sup>4</sup>. Using the well-established TLC, they reported a high prevalence of *pressure of speech* (35%), *tangentiality* (33%), *circumstantiality* (32%) and *self-reference* (28%) in these participants. It is worth mentioning that volunteers had been recruited through advert from local communities (e.g. pubs, libraries, supermarkets, etc.) and had been all screened to exclude psychotic disorders.

The studies reviewed in this section seem to suggest two tentative conclusions. First, TD is not diagnostic-specific symptom but rather a transdiagnostic phenomenon that can be identified across a range of diagnostic groups. Second, TD is not a specific symptom of psychosis (or schizophrenia) but rather a fairly common phenomenon that can be identified even in non-clinical and healthy individuals. However, these conclusions must be tempered by the very limited epidemiological evidence on TD, and by the absence of studies that address the question of whether there could be a specific taxon of individuals who are likely to show evidence of it. The dearth of studies in this area no doubt reflects the practical difficulty of measuring TD in the large samples that are necessary for these purposes.

#### **2.2.4 ‘Word salad’ or meaningful utterances?**

One aspect of TD that has definitely divided opinions in psychiatry is the issue of its *intrinsic meaning* (Szasz, 1993). Psychiatric terms such as “schizophasia” or “word salad” have often been used in research contexts and clinical settings to describe the unintelligibility of TD or to allude to the *apparent* mixture of confused and random words and phrases in the speech of psychotic individuals. In fact, much of the

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<sup>f</sup> by eccentric the authors meant individuals who were nonconforming, creative, strongly motivated by curiosity and intelligent.

research carried out to date has used a *deficit framework* with the underlying assumption that TD is a consequence of a genetically-determined neuropathological deficit in language-related areas in the brain (Horn et al., 2009; Kircher et al., 2001; Shenton et al., 1992). However, an alternative perspective is to look at TD as an incoherent way that individuals use to express something that is meaningful to them (Bentall, 2003), a psychological need (Jung, 1907) or something that is relevant to their *being-in-the-world* (Laing, 1960).

Cameron (1938) was the first researcher to inadvertently highlight the issue of personal meaning in TD by coining the term, *interpenetration of themes*. The term referred to the systematic observation of a tendency of thought-disordered participants to incorporate their own preoccupations and worries in the context of experimental tasks (Cameron, 1939). However, it was not until Martin Harrow and colleagues' work at the University of Chicago that the central issue of meaning in TD was addressed (Harrow, Lanin-Kettering, Prosen, & Miller, 1983; Harrow & Prosen, 1978, 1979; Harrow & Quinlan, 1985; Lanin-Kettering & Harrow, 1985; Marengo & Harrow, 1985).

The underlying hypothesis in Harrow and colleagues' work was that thought-disordered participants were hard to understand not because cognitions and speech were randomly assembled but instead because patients' personal concerns and worries *blended in* with the context of conversation, leading to idiosyncratic and at times bizarre associations. Harrow and colleagues (1983) coined the word *intermingling* to describe this phenomenon, which they defined as:

“A tendency to blend material that comes from one's own past or recent experiences into current thinking and communication (...) to be considered

intermingling, the personalized material must enter into the response in a manner that makes it appear strange or inappropriate to current speech situation” (p. 355).

This concept is not at all different from Cameron’s concept of interpenetration of themes. However, Harrow suggested that the *bizarre* and *idiosyncratic* verbalisations of patients were also explained by an “impaired perspective” i.e. an inability to view communication from the perspective of the listener, leading to a failure to comply with both the listener’s communicational needs and with consensual standards of communication (Harrow, Lanin-Kettering, & Miller, 1989).

In one of their studies using their own scoring system (BIT see Table 2; Marengo et al., 1986), Harrow and colleagues (1983) reported that 68% of the psychotic patients in their sample produced verbalisations that were influenced by the intermingling of decontextualized personal material. Moreover, the majority of these patients continued to show adherence to the original question i.e. the participant did not lose the cognitive set completely despite some loss of goal-directed thinking. The most surprising finding in this study came a week later when researchers conducted a standardised interview with participants with the aim to clarify some of their *bizarre* and *idiosyncratic* responses. Interestingly, Harrow and colleagues reported that the *bizarreness* of these thought-disordered utterances was only apparent; when participants provided more context and information about the intended meaning behind these utterances these were actually comprehensible. The authors concluded:

“In most instances in which there was a question about the link between to externally disparate ideas, after the subject supplied additional material, it was

fairly easy to see how, when looked at from the subject's point of view, the two themes were closely related" (p. 360).

The findings reported by Harrow and colleagues are consistent across their published studies (Harrow et al., 1983; Harrow & Prosen, 1978, 1979). For example, in a later study, Harrow and colleagues (2003) reported that a significant percentage of psychotic participants who showed evidence of TD when interpreting proverbs had also shown high levels of intermingling of personal concerns during the object sorting task in line with Cameron's concept of interpenetration of themes.

In another study, Harrow and colleagues (2000) reported that thought-disordered participants showed a tendency to stray from the context of conversation but they rarely ignored the context completely. In their discussion of these findings, the authors suggested that, during heightened cognitive arousal, personal material from the patient's affective inner life becomes salient, merging and interfering with the guiding set and driving away the attentional focus of the individual and their associative processes, consequently leading to TD.

This account puts a different twist into earlier conceptualizations of TD as it introduces the notion of personal symbolism and meaning, suggesting that the apparent *unintelligibility* of TD, often seen as evidence of brain pathology, could instead reflect personal meanings that are not immediately accessible to the listener. In line with this idea, Swartz and Swartz (1987) have shown that, through the use of discourse analysis and adequate contextualization, the apparently senseless speech of psychotic individuals can become understandable.

This different interpretative stance is important when looking back at early studies which reported that patients made more bizarre associations (Schwartz, 1982)

and at more recent studies which have suggested disorganisation of the semantic networks in TD (Goldberg & Weinberger, 2000; Goldberg et al., 1998). On this issue Beck and colleagues (2009) have suggested that TD represents an encroachment into the patient's discourse of *hypersalient cognitions* that are timely and emotionally significant to the patient:

“We argue here that some of these associations could actually have some psychological significance to the patient (...) We predict that, in specific instances, those with formal thought disorder will experience increased spreading of activation to nodes that are based on more personal associations” (Beck et al., 2009, p. 170).

Maher (1983), one of the early proponents of what came to be known as the dyssemantic hypothesis of TD, also suggested that, over time, the repeated experience of intrusive thoughts could lead to the remodelling of associations between concepts, thereby explaining the idiosyncratic quality of TD. Although largely untested, this kind of model of TD has interesting and constructive implications for both clinical communication with patients and psychotherapy (Beck et al., 2009; Galletly & Crichton, 2011).

### **2.2.5 ‘Hot’ and ‘cold’ thought disorder?**

Stress and negative affect have long been acknowledged as important factors in both the aetiology and course of psychotic disorders (Nuechterlein & Dawson, 1984; Zubin & Spring, 1977; Walker & Diforio, 1997) and there is now a wealth of published work suggesting that psychotic patients are especially reactive to daily-life stresses



(Myin-Germeys & van Os, 2007), familial expressed emotion (Hooley, 2007), and life events (Beards, et al., 2013), and that such reactivity may predict subtle momentary variations in psychotic symptoms in the flow of everyday life (Myin-Germeys, Delespaul, & Van Os, 2005). Concurrently, there is also a considerable volume of published work documenting the effect of arousal and negative affect on communication and TD (i.e. affective reactivity of speech, Docherty, et al., 1994; Docherty, Sledge, & Wexler, 1994; Mohagheghi, Farnam, Farhang, & Bakhshipoor, 2012; Rubino et al., 2011), although such effect has not always been acknowledged (Chapman & Chapman, 1973).

In one of the first studies ever published to test the effect of emotional salience in patients' communication, Docherty and colleagues (1994) tested several participants diagnosed with schizophrenia on both TD and *unclear linguistic references* (Halliday & Hasan, 1976). In order to test the effect of emotional salience, participants were interviewed with two counterbalanced 10-minute speech tasks in which they were prompted to talk about "stressful times" and "pleasant, non-stressful times", separately. As predicted, participants diagnosed with schizophrenia displayed significantly more TD and *unclear linguistic references* in the stressful condition. This finding (i.e. affective reactivity of speech) was later replicated by the same authors (Docherty, Hall, & Gordinier, 1998; Docherty, Sledge, & Wexler, 1994).

In probably the first UK study on the impact of negative affect on TD, Haddock and colleagues (1995) tested the same hypothesis as Docherty and colleagues but using a set of carefully developed salient and neutral interviews. The questions in the salient interview had been devised to promote personal disclosure (e.g. "Could you tell me about the most awful thing that someone has done to you?") and the speech samples were scored with the TLC (Andreasen, 1986). As expected,

thought-disordered participants displayed more TD in the salient interview as opposed to non-thought disordered participants who did not show affective reactivity of speech. Using the same methodology, Tai, Haddock and Bentall (2004) reported the same affective reactivity of speech in participants diagnosed with Bipolar Affective Disorder.

With the aim of testing the physiological expression of affective reactivity of speech, Docherty and Grillon (1995), measured the *startle response* and its association with affective reactivity of speech in participants diagnosed with schizophrenia. For the purpose, the researchers administered an *acoustic startle test* and measured eye blink using *orbicularis oculi electromyography* (EMG). During this task, participants were exposed to 40 milliseconds duration bursts of white noise [105 dB(A)] with some of the trials being preceded by a 30 milliseconds duration prepulse stimulus [75 dB(A)]. Interestingly, the researchers reported a significant interaction between high startle response and *unclear references* per clause of speech in the stressful speech condition. In other words, participants who displayed more reactivity of speech also showed higher amplitude of startle reflex.

In a second study, Docherty and colleagues (2001) tested the same idea but now with a more robust methodology. They reported that participants who displayed more affective reactivity of speech also displayed larger amplitude in the initial startle. However, they also showed more habituation and no relationship was found with prepulse inhibition (prepulse inhibition refers to a neurological phenomenon in which a weaker acoustic pre-stimulus [prepulse] inhibits or weakens the reaction of the individual to a stronger subsequent acoustic startling stimulus [pulse]). Of interest is that participants displayed more communication disturbances in the salient speech task and reported experiencing more subjective and momentary stress.

Interested in exploring how affective reactivity of speech is associated with expression of emotion, Cohen and Docherty (2004) assessed participants diagnosed with schizophrenia for *deficit syndrome* and tested them on affective reactivity of speech using the speech tasks described above. Although *deficit syndrome* is far from a consensual concept it is generally accepted to refer to patients with restricted expression of affect, diminished subjective emotional experience (anhedonia), poverty of speech, demotivation and social withdrawal (Carpenter, Heinrichs, & Wagman, 1988). Statistical analyses revealed that *deficit*, *non-deficit* and comparisons all displayed some degree of affective reactivity of speech. However, *non-deficit* participants showed significantly more affective reactivity than the other two groups. Further analysis, revealed that severity of *deficit* symptoms was significantly and negatively correlated with affective reactivity of speech.

The findings supporting the affective reactivity of speech in some participants diagnosed with schizophrenia have since been replicated in participants who score highly on disorganised schizotypy (Kerns & Becker, 2008; Minor & Cohen, 2010). In one of these studies, Kerns and Becker (2008) collected positive and negative speech samples using a modified version of the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) which were scored with the CDI (Docherty, 1996a). Kerns and Becker also tested participants on emotional ambivalence (SAS; Kwapil, Mann, & Raulin, 2002) and working memory (N-back). As expected, the participants with high scores on disorganised schizotypy displayed significantly more communication disturbances in the negative speech task and these disturbances were associated with poor performance on the working memory task. More importantly, emotional ambivalence in the disorganised group was strongly associated with

increased communication disturbances suggesting some potential association between difficulties with emotional processing and affective reactivity of speech.

In another study, Minor and Cohen (2010) tested affective reactivity of speech in a sample of psychometrically-defined schizotypal participants using pleasant and unpleasant photographs (IAPS, Lang, Bradley, & Cuthbert, 1999). In this study, participants had to produce speech whilst viewing the photographs. The authors reported a significant correlation between CDI scores in response to stressful photographs and disorganised schizotypy.

Finally, Docherty and Hebert (1997) coded the speech samples of participants diagnosed with schizophrenia using both the CDI (Docherty, 1996a) and the TLC (Andreasen, 1986). As expected, the researchers reported a significant increase in positive TD during the interview about negative autobiographical memories, but not on negative TD, replicating earlier findings (Docherty, et al., 1994). Interestingly, *vague* and *confused references* as well as *ambiguous word meanings* displayed high reactivity across the speech task but *missing information*, *wrong word references* and *structural unclarities* did not. In later replication of this study, Docherty, Hall, and Gordinier (1998) reported again similar findings with *vague* and *confused references* and *ambiguous word meanings* increasing significantly in response to negative affect in participants diagnosed with schizophrenia.

It is now reasonably clear that the negative affect elicited by the disclosure of personal and salient memories seems to have a detrimental impact on the quality of communication (Burbridge & Barch, 2002). This effect does not seem to be generalizable to positive affect, which seems to improve communication in individuals diagnosed with schizophrenia (Cohen & Docherty, 2005). However, the

question of how *negative affect* may affect communication, speech and cognition is a different matter altogether.

In a study with healthy volunteers, Burbridge, Larsen and Barch (2005) reported that increased linguistic reference errors during a salient interview was significantly associated with increases in heart rate and in frequency of nonspecific skin conductance responses. Using a biological framework, they suggested that discussing emotionally-laden topics could lead to increased autonomic arousal which would in turn lead to changes in dopamine and norepinephrine, which are known to modulate activity in the prefrontal brain regions such as the dorsolateral prefrontal cortex (dlPFC). These brain areas are known to support working memory, which is a necessary resource for monitoring *discursive referents* during conversation.

Using a more cognitive framework, Beck and colleagues (2009) have suggested that communication disturbances could result from the cognitive load (e.g. negative automatic thoughts) imposed by an expectation of rejection (i.e. allocation of limited memory and attentional resources to interpersonal cues) on an already depleted cognitive system, leading to disorganisation of speech. If this is indeed the mechanism, it could be further exacerbated by the fact that thought-disordered participants are aware of their difficulties with communication (McGrath & Allman, 2000).

This account suggests the need for a more interpersonal framework for understanding thought and communication disturbances. Consistent with this approach, social-evaluative threats are known to be the most powerful predictors of cortisol and adrenocorticotrophic hormone (ACTH) changes in healthy individuals (Dickerson & Kemeny, 2004) and it seems likely that the same is true for psychotic patients (Jones & Fernyhough, 2007). For example, St-Hilaire and Docherty (2005)

reported that participants diagnosed with schizophrenia who displayed more affective reactivity of speech tended to report significantly more *difficulties relating to others* and *fear of social relationships* in a work disruption checklist.

In an earlier study, Shimkunas (1972) had reported that participants diagnosed with schizophrenia produced significantly more bizarre, illogical and tangential verbalisations when asked to make disclosures of personal significance. Shimkunas interpreted these findings as evidence that thought-disordered participants avoid personal disclosure as a psychological manoeuvre to distance themselves from an environment which has been invalidating. Although highly speculative, such account is consistent with more recent findings from Grant and Beck (2009) who reported that *evaluation sensitivity* (i.e. dysfunctional beliefs about social acceptance) moderated the relationship between performance on neurocognitive tests and TD in psychotic patients and Dozier and Lee (1995) that reported a significant association between deactivating attachment strategies<sup>5</sup> (AAI; George, Kaplan, & Main, 1985) and TD in participants diagnosed with schizophrenia and BPAD. To the best of our knowledge, the hypotheses that TD could represent a psychological defence against closeness (Haley, 1959) or anxiety (Sullivan, 1964) have never been directly tested.

Although not addressing this issue directly, Seghers and Docherty (2009) tested the role of stress sensitivity in affective reactivity of speech using a more interpersonal framework, by including patients' significant others (SO). At baseline, participants diagnosed with schizophrenia were tested on sustained attention (Degraded-stimulus Continuous Performance Test; Nuechterlein, Parasuraman, & Jiang, 1983), immediate auditory recall (Digit Span Task; Wechsler, 1974), organisational (Trail Making Test-B; Reitan & Davidson, 1974) and abstract-

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<sup>5</sup> Deactivating strategies are assumed to be employed when the person perceives *proximity-seeking* as dangerous and help maintaining psychological distance, suppress attachment-related needs, avoid intimacy, emotional involvement and self-disclosure (Shaver & Mikulincer, 2007).

conceptual sequencing (Shipley Institute of Living Scale; Shipley, 1940), variables that have been found to be associated with communication disturbances (Docherty, Hall, Gordinier, & Cutting, 2000). Along with the neurocognitive tests, participants were assessed for depression and *stress sensitivity* (composite score of the SO expressed emotion and participants' subjective rating of stress when in the presence of SO). The participants were then interviewed on negative and positive topics and speech samples were coded using the CDI (Docherty, 1996a). Curiously, none of the neurocognitive variables were found to be significantly associated with affective reactivity of speech but depression was. More importantly, *stress sensitivity* was strongly correlated with affective reactivity of speech and it was found to mediate the relationship between depression and affective reactivity of speech. In the discussion of the findings, authors write:

“We suggest that as speech-critical neurocognitive functions such as attention and memory are temporarily worsened by the individual's stress response (which may in turn be moderated by individual-level factors such as depression and threat appraisal), gross levels of speech disturbance are increased. Because this kind of neurocognitive mediation would be limited to the proximal, ‘in-the-moment’ stress response, baseline measures of neurocognitive functioning may not predict speech reactivity to the extent that they have been shown to predict non-reactive impaired communication.” (Seghers & Docherty, 2009, p. 101).

The results from these studies and other studies (Rhinewine & Docherty, 2002; Docherty, Grosh, & Wexler, 1996) led Docherty (1996b) to suggest that

*affective reactivity* could be a process discriminator of some patients diagnosed with schizophrenia involving *catecholaminergic overactivity* (e.g. through long-term potentiation of specific stress reactive dopaminergic neural pathways) and specific deficits at the level of inhibitory mechanisms such as *sensory gating* and *habituation*.

It seems logical to conclude that affective reactivity of speech (like TD) is a transdiagnostic phenomenon that is understandably worsened by contextual or “in-the-moment” variables. Moreover, the static assessment of “cold” and out of context cognition is likely to carry important shortcomings (Gjerde, 1983). It is not surprising that TD in psychotic patients seems to be more prominent upon admission to mental health ward when the patient is experiencing a crisis (Marengo & Harrow, 1987). In fact, the dynamic nature of TD and communication disturbances may well be dependent on dynamic, relational and emotional aspects of the individual’s life or, in other words, dependent on the immediate interpersonal environment of the individual.

## **2.3 Neurocognitive mechanisms and TD**

Cohen and Servan-Schreiber (Cohen, Barch, Carter, & Servan-Schreiber, 1999; Cohen & Servan-Schreiber, 1992) proposed one of the most influential neuroscientific theories of schizophrenia, cognitive disorganisation and TD. The theory had the merit of bringing together both the biological level of analysis and information processing deficits.

At the centre of their theory was the idea that patients show a core difficulty representing contextual information (i.e. difficulty integrating contextually appropriate stored information with information from the environment and on-going behavioural responses). This difficulty was believed to be due to disruptions in the



context-processing module in the pre-frontal cortex (which supports both the representation and maintenance of context information, buffering it against interference from task-irrelevant stimuli and maintaining it available to be updated as required by the changing environment).

This final common pathway was assumed to comprise two cognitive processes, working memory (WM) and inhibitory control, as both are crucial in the generation and maintenance of an online *internal representation of context* during goal-directed activity. The internal representation of context referred to relevant contextual information (declarative or non-declarative) about the environment that is crucial to regulate behavioural responses and to inhibit the interference and intrusion of context-irrelevant information.

In a later study carried out to test their theory, Cohen and colleagues (1999) reported that difficulties with internal representation of context were significantly associated with conceptual disorganisation, rather than schizophrenia as a diagnostic group, and more recently Roesch-Ely and colleagues (2010) have replicated these results showing a significant association between degradation of the context-module and TD. It is worth mentioning that in both studies the hypotheses were tested using neuropsychological tests (e.g. Stroop test, Continuous performance test, or the lexical disambiguation task) that require the efficient use of context.

Consistent with Cohen and Servan-Schreiber's model are findings from studies that document that thought-disordered participants are less sensitive to context making more pragmatic, semantic and syntactic violations in word-monitoring paradigms (Kuperberg, McGuire, & David, 1998, 2000) and also studies that have documented that thought-disordered patients have difficulty inhibiting the intrusion of irrelevant material (Brébion, Gorman, Amador, Malaspina, & Sharif, 2002; Fridberg,

Brenner, & Lysaker, 2010; Subotnik et al., 2006; Torres, O’Leary, & Andreasen, 2004).

The context-module is believed to be supported by the *dorsolateral prefrontal cortex* (dlPFC) that is dependent on phasic dopamine activity to regulate signal-to-noise ratio in the pre-frontal cortex modulating the gate between prefrontal context processing module and the repertoire of learned behavioural contingencies (Barch, Carter, MacDonald, Braver, & Cohen, 2003; Cohen, Barch, Carter, & Servan-Schreiber, 1999; Cohen & Servan-Schreiber, 1992; Goldman-Rakic, 1994; Goldman-Rakic, 1987; Servan-Schreiber, Cohen, & Steingard, 1996).

Interested in testing the role of the dlPFC and working memory in TD, Perlstein, Carter, Noll and Cohen (2001) scanned participants diagnosed with schizophrenia and healthy comparisons during a n-back task using *functional magnetic resonance imaging* (fMRI). The analysis of the imaging data revealed that disorganisation symptoms in patients were significantly associated with changes in signal intensity in the right dlPFC as a function of working memory load.

The results of this study are strengthened by the results of a recent meta-analysis documenting a robust association between abnormalities in the activation of the dlPFC in fMRI studies and cognitive disorganisation in patients (Goghari, Sponheim, & MacDonald, 2010). Further evidence to support the role of the context-module in TD comes from semantic priming studies that have reported that patients show a hypopriming effect at long stimulus onset asynchrony (Minzenberg, Ober, & Vinogradov, 2002; Pomarol-Clotet et al., 2008). This is relevant because in lexical decision tasks (which we will discuss below), a long stimulus onset asynchrony is assumed to facilitate the generation of a contextual expectancy through a more controlled set of cognitive processes. The evidence for a hypopriming effect is

suggestive that these “contextual” processes are not being primed. Moreover, *event-related potential* (ERP) studies have documented an attenuation of the N400 component in thought-disordered participants (Andrews et al., 1993; Debruille, 2007; Ditman & Kuperberg, 2007; Kostova et al., 2005; Kreher, Goff, & Kuperberg, 2009; Mohammad & DeLisi, 2013; Sitnikova, Goff, & Kuperberg, 2009). The N400 is a negative-going wave that peaks around 400 milliseconds post-stimulus onset and that is thought to reflect general activation of the comprehension network. In semantic priming studies, the presentation of incongruent primes has been found to increase the magnitude of the wave.

### **2.3.1 Executive ability**

One of the theories of TD that has been subjected to more intensive research, and that overlaps greatly with the work on context-representation, is the dysexecutive hypothesis of TD. In a seminal paper, McGrath (1991) developed probably one of the most comprehensive and detailed accounts of the potential role of the executive function in TD. He suggested that different TD symptoms could be explained by four different deficits, namely:

1. A failure to generate a set;
2. An inability to change set;
3. A failure of planning and editing; and,
4. A failure to monitor errors.

This framework produced several testable hypotheses regarding the role of executive function in TD. For example, perseveration could potentially be explained

by difficulties changing set whereas failure to generate a set could potentially explain instances of poverty of speech and poverty of content of speech (by set, we mean cognitive processes, which are not necessarily conscious, that guide our information processing and our behavioural responses). These cognitive deficits were suggested to be associated with frontal lobe pathology but had little specificity as they could also include difficulties with general cognitive control, working memory, cognitive flexibility, abstract reasoning, concept-formation, decision-making or planning of discourse.

In probably the first quantitative review that tested the role of executive functions in TD, Kerns and Berenbaum (2002) reported an overall effect-size of medium strength ( $r = .36$ ) from 26 studies. However, the studies included used a variety of cognitive tasks ranging from Wisconsin card sorting test (WCST) to fluency tasks or saccadic eye tasks. Also, across the studies TD was assessed with different methodologies ranging from TD-related measures (BIS; Harrow & Quinlan, 1985; Marengo et al., 1986) to communication-based methodologies (CDI; Docherty, 1996) making the findings difficult to interpret.

In another relevant meta-analysis, Nieuwenstein, Aleman and De Haan (2001) tested the association between three-symptom dimensions (positive, negative and disorganisation; Liddle, 1987) and performance on two executive tasks, WCST (a neuropsychological test that measures cognitive flexibility by asking the participant to match stimulus cards with different colours, forms or numbers according to different principles) and the *continuous performance test* (CPT; a neuropsychological test that measures sustained and selective attention by asking the participant to identify patterns of sequentially presented visual stimuli; Nuechterlein & Dawson, 1984). Interestingly, the correlation between performance on the CPT and cognitive

disorganisation was non-significant whereas number of perseverations on the WCST was only modestly correlated with cognitive disorganisation ( $r = .25$ ) and with negative symptoms ( $r = .27$ ). Again, one should be cautious interpreting the findings given that cognitive disorganisation is a symptom-dimension extracted from a general measure of psychopathology (e.g. PANSS) that includes a range of symptoms other than TD.

In a more recent meta-analysis, Dibben and colleagues (2009) looked at the association between the same dimensions but including studies that have used different neurocognitive tasks ranging from phonological fluency to the WCST. In their analysis, the authors identified 40 studies with usable data on the relationship between cognitive disorganisation and executive ability. The overall effect-size was quite modest ( $r = -.17$ ). On the sub-analysis by neurocognitive task, the strongest association with cognitive disorganisation was performance on *trails making test* (TMT-B is a neuropsychological test that measures attention, speed of processing and executive function by asking the participant to accurately connect, as fast as possible, a set of dots; Reitan, 1958), the *Hayling test* (this neuropsychological test measures executive function by asking participants to complete sentences, the second part of the test the participant has to use nonsense ending words, Burgess & Shallice, 1997) and the *Stroop test* (this test measures executive function by asking participants to identify congruent and incongruent stimuli, for example the word "red" printed in blue, the dependent variable is both the reaction time and the number of errors, Stroop, 1935). But again, the negative dimension was also found to be significantly associated with poor performance on executive tasks. More importantly, authors reported that the association of cognitive disorganisation and executive ability seem to be significantly

moderated by variables such as duration of illness, treatment status and more importantly IQ.

In a larger review on neurocognition and symptom-dimensions, Dominguez and colleagues (2009) tested the associations between a range of cognitive domains from IQ to visual learning and symptoms. Again, the authors reported significant correlations between poor performance on the different cognitive tasks and both the negative and disorganisation dimensions. However, whilst the negative dimension was more strongly correlated with verbal fluency tasks, the disorganisation dimension was strongly correlated with reasoning and problem-solving tasks and attention/vigilance tasks.

Finally, Ventura and colleagues (2010) meta-analysed 104 studies and found again a moderate association between disorganisation-symptoms and neurocognition ( $r = -.23$ ;  $p < .01$ ). The neurocognitive domains most strongly correlated with disorganisation were speed of processing ( $r = -.26$ ;  $p = .01$ ), attention/vigilance ( $r = -.25$ ;  $p < .01$ ) and reasoning and problem solving ( $r = -.24$ ;  $p < .01$ ).

Although informative, the findings from these meta-analyses need to be interpreted with caution as they included studies that did not measure TD individually (the disorganisation-dimension derived from general psychopathology scales include symptoms other than TD). Also, as mentioned earlier, TD is a multidimensional construct with categories that represent opposite phenomena (e.g. poverty of speech and pressure of speech). Moreover, McGrath's framework suggested more specific associations between specific deficits and specific symptoms (e.g. a failure to generate a set was suggested to be associated with poverty of speech, whereas an inability to change set was suggested to be associated with perseveration, McGrath, 1991) .

Several authors have carried out rigorous studies testing the relationship between executive function and TD. Kerns and Berenbaum (2003) tested participants diagnosed with schizophrenia using a range of different cognitive tasks and reported significant and robust associations between communication disturbances (CDI; Docherty, 1996) and performance on both the 2-back task and on the Sternberg probe recognition with interference task (in this task, participants have to identify previously presented stimuli and interference is calculated from the difference in reaction time between the recognition of recent and less recent items, Jonides, Smith, Marshuetz, Koeppe, & Reuter-Lorenz, 1998). In the same study, the authors also reported that the interaction between poor processing of context (N-back task) and high interference (Sternberg task) was highly predictive of communication disturbances.

Finally, Barrera, McKenna and Berrios (2005) carried out a rigorous study of executive ability and TD with participants carefully matched for general intelligence. They reported that thought-disordered participants had a significantly poorer performance on the *Hayling test* (Burgess & Shallice, 1997), the *modified six elements test* (This is a neuropsychological test of executive function in which the participant is asked to complete six different tasks, e.g. picture naming, arithmetic, etc., in 10-minutes, Wilson, Alderman, Burgess, Emslie, & Evand, 1996), the *Brixton test* (This test is a visuospatial sequence task that measures executive function by asking the participant to identify rules in sequences of stimuli, Burgess & Shallice, 1997) and the *cognitive estimates test* (The CET is a test that measures executive function by asking the participant to answer a set of questions that require deductive reasoning, Shallice & Evans, 1978) when compared with both controls and non-thought disordered participants. These two latter studies had the advantage of being carefully designed and executed and they have provided some support for both the

dysexecutive hypothesis of TD and the degradation of the internal representation of context.

It seems reasonable to suggest that difficulties with inhibitory control (Kerns & Berenbaum, 2002) may explain the intrusion and *blending in* of decontextualised personal worries and concerns in the speech of thought-disordered participants. However, deficits in executive function are found across a range of psychological and medical disorders where TD is not a prominent feature. Also, in many studies general intellectual functioning and symptom co-morbidity have not been accounted for, making it difficult to assess the specificity of executive function.

## **2.4 Psychological mechanisms**

Three specific cognitive mechanisms have often been evoked in attempts to explain TD, namely: semantic hyperpriming, poor internal source monitoring and poor 'theory-of-mind'.

### **2.4.1 Semantic hyperpriming**

The early empirical work on TD was influenced by the concept of *loosening of associations* (Bleuler, 1911; Schwartz, 1982). The concept was meant to represent a fundamental deficit at the level of *associative processes* or, to use more contemporary terminology, at the level of the semantic networks (McKenna & Oh, 2005; Pomarol-Clotet, et al., 2008; Spitzer, 1997). According to Bleuler, one manifestation of this core deficit was the indirect, mediated and oblique associations that prevailed in the patients' thinking and speech:



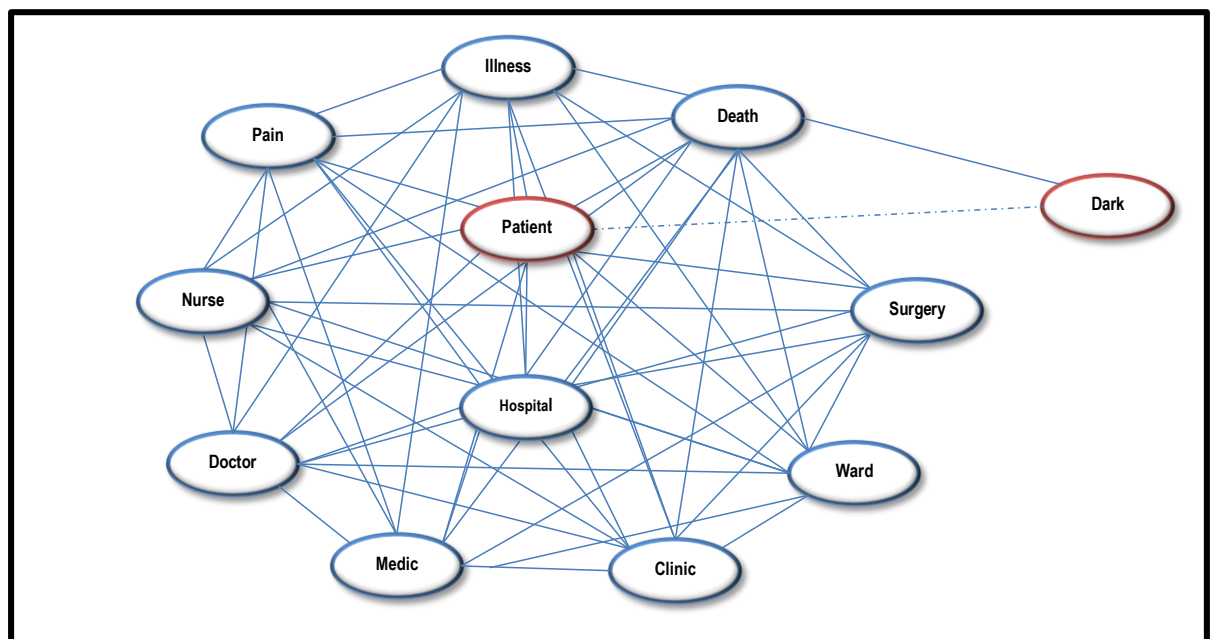
“In this malady the associations lose their continuity. Of the thousands of associative threads, which guide our thinking, this disease seems to interrupt, quite haphazardly, sometimes single threads, sometimes a whole group, and sometimes even larger segments of them. In this way, thinking becomes illogical and often bizarre. Furthermore associations tend to proceed along new lines.” (Bleuler, 1911, p. 14).

The concept of *loosening of associations* motivated many of the early word association studies (Kent & Rosonoff, 1910; Schwartz, 1982) in which the common reported finding was that patients made more idiosyncratic or less normative associations to target words (Johnson, Weiss, & Zelhart, 1964; Kent & Rosonoff, 1910; Moran, 1953). These early studies influenced other theories and paradigms (e.g. Cameron, 1938) and appeared to be consistent with results of linguistic studies that reported that speech of patients diagnosed with schizophrenia was less predictable than that of healthy individuals (Maher, et al., 1988; Manschreck, et al., 1979; Ragin & Oltmanns, 1983). Despite having been heavily criticized for their methodological shortcomings and equivocal findings (*see* Schwartz, 1982), these studies have left a significant mark in the field (Goldberg & Weinberger, 2000).

Nowadays the dyssemantic hypothesis is tested with more sophisticated methodologies and remains has probably the most influential theory in the field (McKenna & Oh, 2005). Over the last 30 years, one approach has been to use *semantic priming* paradigms (Collins & Loftus, 1975), leading to the publication of numerous studies (Kerns & Berenbaum, 2002; Minzenberg, Ober, & Vinogradov, 2002; Pomarol-Clotet, et al., 2008). The core idea behind this line of investigation is that TD is a consequence of an enhanced level of activation in semantic memory

during communication (Maher, 1983; Spitzer, 1997) and that such activation leads to the inadvertent disruption of the semantic associative networks that underlie the speech of the patient.

In these theories and experiments, semantic memory is conceptualized as a network of interconnected nodes (semantic concepts) with different associative strengths (Figure 1). The conceptualized space between nodes is supposed to reflect an associative strength and the semantic relatedness of the two concepts. During everyday conversation, on a moment-to-moment basis, several concepts are assumed to be *primed* through an unconscious mechanism called *spreading of activation*. This mechanism is assumed to facilitate lexical access by lowering the threshold of activation to related and neighbouring nodes in the *semantic space*.



**Figure 1** - Representation of a semantic network.

For example, in Figure 1 the activation of the node “Patient” would automatically lower the threshold of activation to semantically related nodes such as “Hospital” and “Illness”. This automatic activation of the semantic nodes is assumed to be pre-attentional process and to decay after a few hundred milliseconds before being taken over by more controlled cognitive processes (Collins & Loftus, 1975; Spitzer, 1997). Semantic priming is the facilitation effect that occurs when the threshold of activation to a node is lowered (this effect can also have an inhibitory component by delaying access to unrelated words).

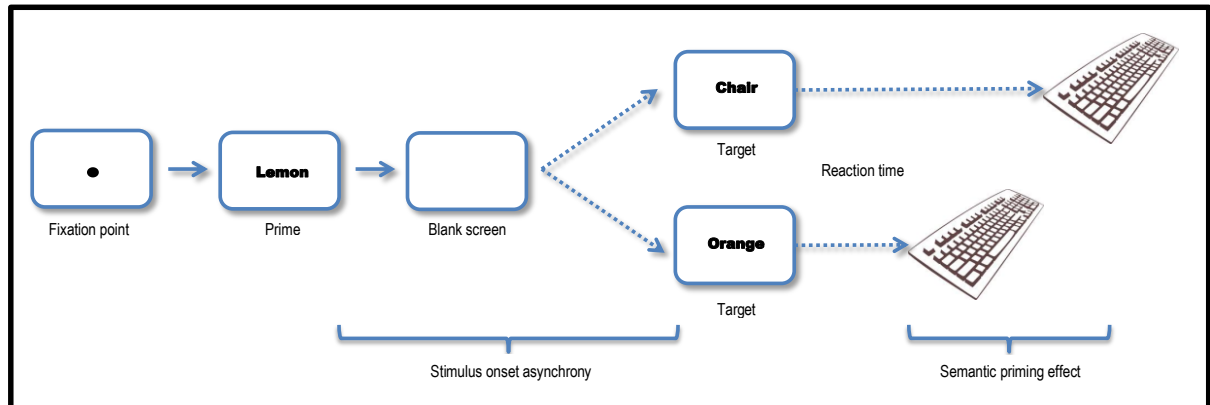
This effect has commonly been tested with lexical decision tasks (LDT *see* Figure 2). In this task, the participant is initially presented with a prime-word and later with a target-word. The different prime and target-words are assembled on the basis of their strength of association through the use of standardised norms. So for example, in the congruent condition, the participant is presented with a prime, which is semantically related to the target-word (e.g. lemon – orange) as opposed to the incongruent or unrelated condition (e.g. lemon – chair). The semantic priming effect is the facilitatory effect caused by the presentation of a semantically related prime before the target, which is measured in terms of reaction-time gain between the congruent and the incongruent conditions<sup>6</sup>. So for example, if the prime is strongly associated with the target word, one would expect a quicker reaction time in the recognition of the target. On the other hand, if the prime is not related to the target one would expect a longer reaction time.

One important variable in the LDT studies is the *stimulus onset asynchrony* (SOA). The SOA reflects the interval in milliseconds between the presentation of the prime and the target. Shorter SOAs ( $\leq 400$  milliseconds) are generally assumed to

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<sup>6</sup> Some studies have opted to also use a neutral condition. In these studies, semantic priming is calculated not just by the facilitatory effect (RT congruent – RT neutral) but also by the inhibitory effect calculated by subtracting the reaction times in the incongruent and neutral condition.

reflect more automatic and pre-attentive processes (i.e. spreading of activation) whereas longer SOAs (> 400 milliseconds) are believed to reflect more controlled processes (with a generation of expectations regarding potential targets).



**Figure 2** - Lexical decision task paradigm.

Several authors have used the semantic priming framework to develop elegant and testable theories of TD (Maher, 1983; Spitzer, 1997; Spitzer, et al., 1993) but overall the results of the studies have been difficult to interpret. Some studies have suggested that thought-disordered participants have an enhanced priming effect (Lecardeur, et al., 2007; Moritz, et al., 2001a; Moritz, et al., 2001b; Moritz, et al., 2003; Safadi, et al., 2013; Spitzer, et al., 1993; Spitzer, et al., 1994; Weisbrod, et al., 1998) and these findings have been replicated with other priming paradigms (Kiefer, et al., 2009; Quelen, Grainger, & Raymond, 2005). However, other studies have suggested normal (Barch, et al., 1996; Besche-Richard, Passerieux, & Hardy-Baylé, 2005; Blum & Freides, 1995) or even decreased priming (hypopriming) in thought-disordered participants (Aloia, et al., 1998; Besche-Richard & Passerieux, 2003; Besche, et al., 1997; Passerieux, et al., 1997).

Mainly on the basis of studies that found evidence of hypopriming, Goldberg and colleagues (Goldberg, et al., 1998; Goldberg & Weinberger, 2000) argued that at the core of TD could be a disorganised semantic network. Although, this hypothesis appears to address the idiosyncratic nature of TD, it is clear that findings have not supported it (Green, Done, Anthony, McKenna, & Ochocki, 2004).

One potential explanation for the conflicting findings becomes clear in an study carried out by Gouzoulis-Mayfrank and colleagues (2003). In this study the authors tested a group of thought-disordered participants shortly after their admission to an acute ward and again 12-16 weeks later. Interestingly, they reported evidence supporting hyperpriming at time 1 (when participants were psychotic and acutely unwell) but not at time 2, suggesting that hyperpriming may be state-dependent, which is consistent with the role of negative affect and emotional salience in TD (Docherty, Evans, Sledge, Seibyl, & Krystal, 1994; Docherty, 1996a; Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995; Tai et al., 2004) which we discussed above.

In a review of the semantic priming studies of TD, Minzenberg, Ober, and Vinogradov (2002) concluded that studies have been often beset by a variety of methodological problems. For example, despite the evidence supporting the multi-dimensionality and orthogonality of TD (Cuesta & Peralta, 1999) some studies have used a single-item rating scale to assess TD (or used arbitrary thresholds for the TD criteria). Also, more important to the argument of specificity, some studies lack direct statistical comparisons between thought-disordered and non-thought-disordered groups and instead report the priming effect comparing the former and healthy controls. Another interesting finding from this review was that some studies seemed

to support an impairment in semantic memory in experiments that favoured more controlled processes (longer SOAs).

Spitzer (1997) suggested that priming effect may be enhanced only for mediated and indirectly associated concepts due to an “unfocused” spreading of activation that reaches farther semantic nodes, hypothesis that has received some support (Barch, et al., 1996; Kischka, et al., 1996). Indirectly and mediated concepts are concepts that are related semantically by a third concept. For example, in Figure 1 the words “Patient” and “Dark” are related through the mediating concept “Death”. Spitzer (1997) argued that such abnormalities in the activation of the semantic space could be modulated by a dopaminergic-driven signal-to-noise ratio.

Pomarol-Clotet and colleagues (2008) published a quantitative review of 36 semantic priming studies published between 1988 and 2007. The pooled effect size for schizophrenia as a group was not significant irrespective of SOA. The subgroup analysis of 18 studies comparing thought-disordered participants and healthy comparisons produced a very small effect size ( $d = .16$ ; 95% CI $[-.01; .31]$ ) that was more pronounced when data was sub-analysed across short ( $d = .25$ ) and long SOA<sup>7</sup> ( $d = -.14$ ). On the analysis of 6 studies that tested indirect semantic priming, the pooled effect size for studies that compared thought-disordered participants and healthy comparisons was .56 (95% CI $[.31; .80]$ ). More importantly, subgroup analysis of 13 studies that compared participants with and without TD revealed a non-significant effect size ( $d = .06$ ; 95% CI $[-.12, .24]$ ) and the comparison between short ( $d = .15$ ) and long SOA ( $d = -.17$ ) was not significant.

On further analysis, age and length of illness were not found to be significant confounders however general slowness was. This is especially important given that

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<sup>7</sup> Short SOA was defined as a prime-target interval of  $\leq 400$  milliseconds and long SOA as  $> 400$  milliseconds.

the priming effect (dependent variable in these studies) is calculated from the differences in reaction times in different experimental conditions. So, if the participant is generally slow, the effect will tend to be inflated (*hyperpriming*). On the specific comparison between thought-disordered participants and healthy comparisons, for which a significant effect size had been reported, general slowness was found to be a significant confounder ( $z= 3.23$ ,  $p= .001$ ) meaning that the slower the participant the bigger the priming effect.

If it is true that the results of Pomarol-Clotet and colleagues (2008) are generally disappointing for the hyperpriming theory of TD it is also true that this hypothesis would still not be sufficient to explain the discourse-level disturbances observed in TD such as poverty of the content of speech or categories such as thought block.

#### **2.4.2 Internal source monitoring**

A psychological variable that has been consistently associated with TD is internal source monitoring (i.e. the ability to discriminate between self-generated speech and verbal thoughts or inner speech; Johnson, Hashtroudi, & Lindsay, 1993), hypothesis that was first suggested by Rochester (1978).

It is easy to understand how a difficulty at the level of internal source monitoring could compromise communication as the speaker would not be able to discriminate between information that was only thought (i.e. rehearsed in inner speech or only planned) and not socially shared with the listener (for the listener it would be like reading a text where relevant words or sentences have been deleted).

Alternatively, the inadvertent verbalisation of inner speech could also be problematic in the communicational context. In the later situation, TD would be equivalent to

listening to one's stream-of-consciousness or to segments of speech in the episodic buffer (where the 'jumbled up' quality of TD could be understood as the agrammatical and condensed nature of inner speech, Bentall, 2003).

Consistent with this idea, Cohen (1978) proposed an interesting model in which TD was suggested to result from a faulty capacity to *edit-out* intruding and de-contextualized ideas that had been inadvertently verbalised. However, the most compelling work on self-monitoring has been carried out with the *source-monitoring* paradigm.

In the first study ever published looking at source-monitoring and TD, Harvey (1985) tested participants diagnosed with schizophrenia and BPAD using two different source monitoring tasks. In one of the tasks (listen-listen), two testers read out loud commonly used words in English in alternating fashion. The participant was later asked to discriminate the source of each word (e.g. source 1, source 2). In the second task (say-think), the participants were presented with words written on cards and instructed to either say them out loud or just imagine that they were saying them out loud. The recognition sheets for both tasks included the target-words plus recognition foils. Harvey reported that the thought-disordered participants diagnosed with schizophrenia performed significantly worse on the internal source monitoring task (say-think) when compared to both BPAD and healthy comparisons whereas the BPAD group seem to be significantly worse at the listen-listen task. Perhaps more importantly, thought-disordered participants showed a recognition bias towards reporting that they had said words when they had in fact they had only thought them (think-report-say errors).

In a second study, Harvey, Earleboyer, and Levinson (1988) used the same source monitoring methodology but in a test-retest design to look at cross-temporal



associations across the variables. Again, Harvey and colleagues reported significant and very strong correlations in the schizophrenia group between think-report-say errors on the internal source monitoring task and positive TD at both time-points. Unexpectedly, the correlations between performance on the internal source monitoring task and negative TD were all non-significant. More importantly, Harvey and colleagues reported that it was the interaction term between say-think discrimination and think-report say errors that best predicted positive TD in the schizophrenia group.

In a third study published by Harvey and Serper (1990) the authors tested again a group of participants diagnosed with BPAD and schizophrenia but this time using several neuropsychological tests along with the internal source monitoring. Again, they reported a very strong association between source recognition errors and positive TD in the schizophrenia group but not in the BPAD group. In a multivariate analysis, the authors reported that it was the scores on the digit-span test with distraction along with the source recognition errors that best predicted positive TD.

Finally, Barch and Berenbaum (1996) tested participants diagnosed with schizophrenia using the internal source monitoring task along with other tasks that tapped onto grammatical-phonological encoding and discourse planning. Consistent with previous findings, they reported a significant correlation between poor source monitoring and derailment and non-sequiturs.

The studies carried out by Harvey and colleagues had the merit of providing the first experimental evidence for the role of internal source monitoring in TD. However, there are different source monitoring paradigms in literature and this is especially relevant given that a recent meta-analysis reported a moderate to strong association between auditory hallucinations and a tendency to attribute self-generated

cognitions to external sources, sometimes known as *external source monitoring* (Brookwell, Bentall, & Varese, 2013). In Harvey and colleagues studies, they had not included an external-internal source-monitoring task to understand if there is some specificity between internal source monitoring and TD.

Using a complex word-association paradigm, Brebion and colleagues (2000) tested participants diagnosed with schizophrenia and healthy comparisons on their capacity to recall the source of presented information. During the word-association task, the experimenter indicated a word category (e.g. fruit) read out loud an exemplar (e.g. banana) and showed a picture to the participant with a second exemplar of the category (e.g. picture of an apple). The participant was then asked to produce a third exemplar (e.g. figs). In the recognition part of the task, the participant was presented with the target words and recognition foils and asked to recall the source of the presented information. Interestingly, the thought-disordered participants made more self-misattributions and fewer other-misattributions than other participants, showing the exact opposite bias of participants who had high scores on auditory hallucinations. Unfortunately, Brebion and colleagues did not include an internal source-monitoring task. However the study had the merit of showing that TD is not associated with an externalizing bias i.e. a tendency to misattribute self-generated material to an external source.

Using a word association paradigm to test external-internal attribution biases, Moritz, Woodward, and Ruff (2003) asked participants diagnosed with schizophrenia and healthy comparisons to produce associations in response to 20 prime words. During the recognition phase, the participants were presented with the (1) prime words; (2) their own self-generated words; (3) new unrelated words; and (4) new words associated with their own self-generated words. The authors reported that TD

was strongly associated with a bias to over-report new words as old words (4) and again, TD was found to be not associated with an externalizing bias.

Interested in studying the specificity of think-report say biases in communication and TD, Nienow and Docherty (2004, 2005) developed and piloted a modified internal source monitoring task. In this task, participants were asked to generate one-word responses to self-evident statements printed on individual cards (e.g. “the opposite to left is \_\_\_\_\_.”). In half of the trials, participants were instructed to verbalize out loud the answer (signalled by a card with the word “answer”) whereas in the remaining trials they were instructed to think about it. Immediately after the task, each participant was presented with a recognition sheet with the target-words and new words and asked to identify if the words had been *said*, *thought* or if they were *new*.

In the first of these studies, the researchers reported an association between poor source discrimination and TD, with thought-disordered participants making more say-think discrimination errors. More importantly, these cognitive biases remained significant even after controlling for intellectual ability and verbal working memory.

In the second study, Nienow and Docherty (2005) repeated the same methodology but this time speech samples were coded for communication disturbances (CDI; Docherty, 1996a). Again, the authors reported a significant correlation between source discrimination, think-report-say errors and the CDI item *missing information references*. Unexpectedly, immediate auditory recall and working memory were unrelated to missing referents.

It is easy to understand how missing referents, a communicational disturbance that has been strongly associated with TD (Docherty, 2005), could easily lead to

breakdown of communication, given that it codes for instances where a reference to information has not been previously presented by the speaker and therefore it is not known to the listener.

In a more recent study, Docherty (2012) tested again participants diagnosed with schizophrenia and healthy comparisons using both an internal and an external source monitoring tasks. Again, the author reported a significant association between missing referents and poor internal source monitoring. More importantly, communication disturbances (and conceptual disorganisation) were not associated with poor performance on either the external source monitoring task or poor performance on neuropsychological tests. Finally, hierarchical regression revealed that performance on the internal source monitoring was a significant predictor of missing referents even after controlling for global level of functioning, immediate and working memory performance and external source monitoring.

What is perhaps most striking about the studies reviewed in this section is the very specific and consistent association between poor performance on internal source monitoring tasks and communicational disturbances and TD. This is especially relevant given that this psychological mechanism has the potential to explain the *jumbled up* quality of TD.

### **2.4.3 ‘Theory-of-Mind’**

A social-cognitive mechanism that has been often associated with TD is ‘theory-of-mind’ (ToM). ToM refers to the ability to represent and infer other people’s mental states (*i.e.* knowledge, desires and intentions) from their behaviour and speech. Such ability is an implicit, online and automatic competency that supports most of our social activities including conversation. For this reason, it is not surprising that ToM

has been often associated with the communicational difficulties that thought-disordered participants experience (Cutting & Murphy, 1988).

It was Frith (1992) who first discussed the potential role of impaired ToM in symptoms such as TD in his much broader neuropsychological theory of schizophrenia. According to the model, thought-disordered individuals had difficulties not just representing and monitoring other people's mental states, knowledge and intentions but also at the level of *willed action* (e.g. poverty of speech, perseveration, etc.) and self-monitoring (e.g. 'editing-out' intrusive ideas, etc.). In Frith's own words:

“My conclusion is that some schizophrenic ‘thought disorder’ reflects a disorder of communication, caused in part by a failure of the patient’s to take account of the listener’s knowledge in formulating their speech (...) The schizophrenic speaker does not take account of the listener’s lack of knowledge, and thus the listener has difficulty in understanding.” (1992, p. 100).

Since Frith's influential book, several reviews (Brüne, 2005; Harrington, Siegert, & McClure, 2005) and meta-analyses (Bora, Yucel, & Pantelis, 2009; Sprong, et al., 2007) have been carried out looking at ToM and social cognition in schizophrenia. For example, in a recent review of the field, Savla and colleagues (2012) reported a very sizable pooled effect-size for studies that have examined ToM in schizophrenia ( $k= 50$ ;  $g= .96$ ; 95% CI[.83; 1.09],  $p< .001$ ) which is consistent with the findings from two quantitative reviews (Bora, Yucel, & Pantelis, 2009; Sprong, et

al., 2007). However, this effect-size is for schizophrenia as a whole and tells us very little about the potential specificity of this deficit in TD.

A few studies have looked at the issue of symptom specificity. Corcoran, Mercer, and Frith (1995) tested participants diagnosed with schizophrenia and controls using the hinting task. In this task, participants are shown 10-vignettes of brief social interactions that end with the protagonist of the story making a veiled speech act ('the hint'). Participants are then asked to make social judgments about the intentions of the protagonist in each brief story and ToM is assessed by the accuracy of these judgments. Interestingly, the authors reported that the worst performers were participants with predominantly negative features and incoherent speech. However, the conclusions were seriously limited by the fact that there were only three thought-disordered participants in a whole sample.

Hardy-Baylé, Sarfati, and Passerieux (2003) proposed a model where ToM deficits and failures at the level of integration of contextual information (Cohen & Servan-Schreiber, 1992) were seen jointly as specific mechanisms in TD rather than a generic global-deficit in schizophrenia. In a series of studies, the research group assessed ToM in participants diagnosed with schizophrenia with predominantly disorganised symptoms (Sarfati & Hardy-Baylé, 1999; Sarfati, et al., 1997a; Sarfati, et al., 1999; Sarfati, Passerieux, & Hardy-Bayle, 2000). In the first of these studies, Sarfati and colleagues (1997a; 1997b) reported that participants diagnosed with schizophrenia with TD (TLC; Andreasen, 1986) performed significantly worse than non-thought disordered participants on a task where they had to choose an answer-card to complete a comic strip. The comic strips represented characters performing very simple actions (e.g. a fisherman looking for bait) and the participant had to

identify the correct card to complete the sequence by interpreting the available social cues.

In a later replication of this study, Sarfati and colleagues (1999) reported basically the same findings but this time using a more robust methodology. Again, participants had to infer the mental state of the character by interpreting the social cues in the comic strip and then choose the correct answer-card to logically complete the strip but this time the study included both a pictorial and verbal versions. Thought-disordered participants performed significantly worse than non-thought disordered participants irrespective of whether the answer-cards were pictorial or verbal. These results were later replicated (Sarfati & Hardy-Baylé, 1999; Sarfati, Passerieux, & Hardy-Bayle, 2000).

Interested in the same issue of symptom specificity, Mazza, De Risio, Surian, Roncone, and Casacchia (2001) tested participants diagnosed with schizophrenia using both first- and second-order false-belief tasks (First-order tasks involved the ability to attribute false beliefs of one person in social interaction, whereas in second-order tasks the participant is required to attribute the false belief of one person based on the thoughts of another). Interestingly, the analysis of the participants' performance revealed that psychomotor poverty was especially associated with underperformance on the first-order false-belief task whereas disorganisation was negatively associated with performance on the second-order false belief task.

Using a different methodology, Langdon and colleagues (2002) tested participants using both a picture-sequencing (which tapped onto 'cause-effect' reasoning or 'false-belief' scripts) and a non-literal comprehension task. They found that positive TD was specifically associated with poor appreciation of irony whereas negative TD was specifically associated with poor understanding of metaphors.

In another study, Brüne (2003) tested participants diagnosed with schizophrenia with predominantly disorganised features and healthy comparisons using a variety of ToM tasks (e.g. first-order and second-order false-belief tasks and sequencing cartoon strips). Overall, the thought-disordered participants performed significantly worse in half of the tasks however these differences disappeared when scores were corrected for IQ. The authors concluded by stating that ToM deficits may be contingent on general intelligence, memory, attention and other cognitive domains.

Greig, Bryson, and Bell (2004) also found a strong association between participants' poor performance on the hinting task (Corcoran et al., 1995) and ratings of TD on the Gorham's proverb test (Gorham, 1956) and the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984). However, this association was non-specific as they also found significant negative correlations between performance on the hinting task and delusions, positive and negative symptoms (PANSS; Kay, Fiszbein, & Opler, 1987).

Zalla and colleagues (2006) tested carefully matched (IQ and duration of illness) participants diagnosed with schizophrenia with and without disorganised symptoms using sequencing tasks that tap onto goal-directed action and metalizing abilities. The scripts ranged from short object-related actions and social scripts (with person-to-person interaction) to false-belief stories and objects or events interacting with each other. They reported that thought-disordered participants made significantly more sequencing errors on all tasks, showing what the authors suggested as general difficulties in inferential reasoning. In other words, the findings seemed to suggest that thought-disordered participants have a more general impaired sequencing ability rather than circumscribed difficulties inferring mental states.



In another study, Pousa and colleagues (2008) tested participants diagnosed with schizophrenia along with controls using both a non-verbal picture sequencing task and second-order ToM tasks. Surprisingly, the authors did not report any significant differences when they compared participants with and without TD. Mo, Su, Chan, and Liu (2008) found again no significant differences between disorganised and other subgroups of patients on first-, second-order false-belief tasks and irony and metaphor comprehension (however, in this study there were only four participants in the disorganised subgroup).

Finally, Abdel-Hamid and colleagues (2009) tested participants diagnosed with schizophrenia using six picture sequencing tasks depicting social scenarios of deception and cooperation followed by questions about mental states of the characters in the cartoon strips. Interestingly, the authors reported a significant correlation between scores on the disorganised factor and poor performance on the ToM task even when they controlled for IQ and perseverative errors on the Wisconsin Card Sorting Task. However, this association became non-significant when authors controlled for planning ability (Zoo map test; Wilson, Alderman, Burgess, Emslie, & Evans, 1996).

More recently, Ventura, Wood and Hellemann (2013) carried out a meta-analysis looking at the association between symptom-dimensions and social cognition. They reported significant correlations between cognitive disorganisation and poor performance on *emotion perception* ( $k=22$ ;  $r=-.32$ ), *social perception* ( $k=7$ ;  $r=-.22$ ) and ToM tasks ( $k=16$ ;  $r=-.32$ ). However, the same pattern of associations was found for negative symptoms, suggesting that specific deficits in social cognition may contribute to TD and cognitive disorganisation but they are not symptom-specific.

## 2.5 Genes, heritability and thought disorder

The hypothesis that TD is genetic has been largely supported with findings from studies that have looked at the co-familiarity of communication disturbances (Docherty, Gordinier, Hall, & Cutting, 1999; Docherty, Gordinier, Hall, & Dombrowski, 2004; Docherty, Miller, & Lewis, 1997; Docherty & Gordinier, 1999; Docherty, Hall, et al., 1998; Docherty, Rhinewine, Labhart, & Gordinier, 1998; Docherty, Sledge, et al., 1994; Docherty, 1993, 1995), vague linguistic references (Docherty, 1995; Harvey, Weintraub, & Neale, 1982), thought disorder (Arbelle et al., 1997; Arboleda & Holzman, 1985; Gooding et al., 2012; Haimo & Holzman, 1979; Hain, Maier, Hoechstjanneck, & Franke, 1995; Harrow & Quinlan, 1985; Johnston & Holzman, 1979; Kinney et al., 1997; Remberk, Namyslowska, & Rybakowski, 2012; Shenton, Solovay, Holzman, Coleman, & Gale, 1989), odd speech (Kendler, McGuire, Gruenberg, & Walsh, 1995) and peculiar word use (Baskak, Ozel, Atbasoglu, & Baskak, 2008). This large body of research has often been interpreted as evidence that subclinical TD in the close relatives of patients represent a penetrant expression of ‘schizophrenia susceptibility genes’ (Levy et al., 2010).

Some authors have studied concordance rates in mono (MZ) and dizygotic (DZ) twins to test the heritability of TD. In these studies, the genetic contribution is inferred from the difference between concordance rates given that MZ twins share the same zygote and 100% of the genetic material and DZ twins only share 50% of the genetic make-up (however, this reasoning contains a crucial fallacy, which is the assumption that MZ and DZ twins are raised in the same way; see *equal environment*

*assumption*, Joseph, 2012, which has recently been questioned on empirical grounds, Fosse et al., 2015).

One of the first twin studies to report relevant data on the heritability of TD was carried out by Slater (1953), who reported a concordance rate of 86% for MZ twins and an equally high concordance rate of 69% for DZ twins. Slater concluded that the high concordance rates in both twin pairs suggested that TD could be dependent on non-genetic factors. In another study, Arnold (1971) reported much higher intra-class correlation amongst MZ ( $r = .79$ ) than DZ twins ( $r = .11$ ). However, in this study Arnold used the *Gottschalk-Gleser Content Analysis Scale* to rate the interviews, making these results extremely difficult to interpret given that the items rate symptoms other than TD (The *social alienation and personal disorganisation scale* includes content categories that measure interpersonal functioning rather than TD, Gottschalk, 1997).

In probably the most robust methodological study to date, Berenbaum and colleagues (1985) tested the heritability of TD using a sample of pairs of MZ, DZ and controls (i.e. unpaired twins) all assessed with the TLC (Andreasen, 1986). Interestingly, the authors did not find any significant differences between MZ and DZ twins, casting doubt on the hypothesis that TD is highly heritable.

In another study, Gambini, Campana, Macciardi and Scarone (1997) tested the heritability of TD using 16 MZ and 9 DZ healthy twin pairs. All participants were tested using the TDI (Johnston & Holzman, 1979) and heritability estimate was reported to approach 80-90%. The findings of this small study are difficult to interpret as the study did not include clinical participants and was carried out under the assumption that TD is highly represented in the general population.

In a later study, Docherty and Gottesman (2000) revisited Berenbaum and colleagues earlier study and coded the twins' speech samples for communication disturbances (CDI; Docherty, 1996). Analysis of the CDI scores of MZ and DZ non-schizophrenic twins again did not reveal any significant differences. It was only the separate analysis of *missing information references* that revealed significant differences (with higher heritability estimates for MZ twins), suggesting that this linguistic disturbance may be under genetic-control.

In another study, Cardno, Sham, Murray and McGuffin (2001) studied heritability across the different symptom dimensions. The analysis using within-pair spearman correlations revealed significant differences ( $p < .0005$ ) between MZ twins ( $r = .63$ ) and DZ twins ( $r = .24$ ) on the disorganisation-dimension (however, the dimension was obtained through factor analysis and included loadings on symptoms other than TD).

Another set of studies looked at the prevalence of TD in non-affected siblings of participants diagnosed with psychotic-spectrum disorders. Rietkerk and colleagues (2008) published a meta-analysis of these studies. In their systematic search, only four studies had usable data for analysis of the disorganisation-dimension (Burke, Murphy, Bray, Walsh, & Kendler, 1996; Cardno et al., 1999; Loftus, DeLisi, & Crow, 1998; Niehaus et al., 2005) and the intra-sibling correlation reported was .28 ( $p < .0001$ ).

Væver and colleagues (2005) carried out a pedigree analysis of 329 relatives of six patients diagnosed with schizophrenia. The pedigrees were identified due to the high prevalence of schizophrenia-spectrum and other psychiatric diagnoses in these families (assumed to be indicative of a high genetic signal). TD in the six pedigrees was assessed with the TDI (Johnston & Holzman, 1979). Interestingly, Væver and colleagues did not find a linear relationship between TD and degree of closeness of

the relative i.e. first-degree relatives of patients and more distant or unrelated relatives had the same degree of TD. Curiously, when Væver and colleagues analysed the data from 61 married/cohabitating couples they found no significant differences between biological and *married in* relatives on the TDI scores. Væver and colleagues interpreted this finding as evidence of *assortative mating*.

Several researchers have used molecular genetics to look at the potential role of genes in TD. This is a challenging field of inquiry because it requires large samples who have been genotyped and assessed for TD, and because, in the absence of theory-led predictions about the role of specific genes, a very high level of significance is required (typically  $p < 10^{-7}$  or even higher) to detect differences because of the high number of genes and the problem of multiple comparisons (Clarke et al., 2011).

Serretti, Lattuada, Lorenzi, Lilli and Smeraldi (2000) reported higher disorganisation and delusion scores in DRD<sub>2</sub> S<sub>311</sub>C heterozygotes (Ser<sub>311</sub>/Cys<sub>311</sub>) when compared with DRD<sub>2</sub> homozygotes (Ser<sub>311</sub>/Ser<sub>311</sub>). Despite potentially interesting, an earlier study carried out by Arinami and colleagues (1994) had not found this association.

Wilcox, Faraone, Su, Van Eerdewegh and Tsuang (2002) carried out a genome-wide linkage scan of psychotic symptoms in sibling-pairs and patients diagnosed with schizophrenia. Chromosomes 6, 9 and 20 were identified as suggestive of linkage to factor reflecting the disorganisation syndrome (6p21 and 6q11.2–6q14.2 and 20q11 and 9pter). DeRosse et al. (2008) identified two *single nucleotide polymorphisms* (SNPs) (*rs1415031* and *rs9446083* located in *BAI<sub>3</sub>* gene) within chromosome 6q associated with lifetime severity of disorganisation symptoms in their cohort.

In a comprehensive review of the field, Levy and colleagues (2010) suggested that anomalies at the level of semantic memory could represent more penetrant pleiotropic expressions of a schizophrenia susceptibility gene. In other words, these 'enriched' traits or *endophenotypes* could have a stronger genetic *signal* than the disorder itself and their higher prevalence could increase the statistical power in linkage analyses. In the same paper, Levy and colleagues suggested the *FOXP2*-*CNTNAP2* as genetic pathway of interest for TD, as this gene is known to play a role in the neurodevelopment of language and speech-related functions. However, this hypothesis is challenged by the observation that TD encompasses a range of oddities other than language-related difficulties (Harrow, et al., 1983; Lanin-Kettering & Harrow, 1985).

Around the same time of Levy and colleagues' paper, Tolosa and colleagues (2010) published an association study looking at 27 SNPs from the *FOXP2* gene. The only significant association discovered was between one SNP (rs2253478) and *poverty of speech*. Although interesting, as we have seen poverty of speech is hardly a good representative of TD.

Wang, Zhang, Liu, Wu and Zeng (2012) carried out the first genome-wide association study (GWAS) on TD in patients diagnosed with schizophrenia using a  $p < 10^{-4}$  genome-wide significance level (it is important to note that the significance levels used in this study were well below the standards mentioned above for GWAS studies). The authors reported no less than 61 different SNPs associated with TD. The most significant association was achieved with rs1783925 within the *PKNOX2*, rs2277644 within the *MYH13*, rs12238738 within *PHF2* and rs17196161 within the *GPC6*. Unfortunately, in Wang and colleagues' study, TD was operationalized as a

binary trait (presence or absence) limiting any firm conclusions given the multi-dimensionality and orthogonality of the construct (Cuesta & Peralta, 1999b).

Finally, Fanous and colleagues (2012) carried out GWAS of different symptom dimensions on data from 2,454 patients diagnosed with schizophrenia. Unexpectedly, no association was found between SNPs and the different symptom dimensions. Using polygenic scores (PGS is a sum of trait-associated alleles across many genetic loci, weighted by effect sizes from a GWAS, Iyegbe, Campbell, Butler, Ajnakina, & Sham, 2014), the authors found a significant association between *polygenic signal* and disorganised symptoms such as TD and bizarre behaviour. However, it is important to note that TD was measured with the Lifetime Dimensions of Psychosis Scale (LDPS; Levinson, Mowry, Escamilla, & Faraone, 2002) which is a scale of general psychopathology. Moreover, the polygenic scores only explained a very small amount of the variance.

Looking at the evidence from the molecular studies reviewed, the most optimistic conclusion is that the genetic component of TD is still to be found. Generally speaking, studies have found conflicting findings and replications are in great demand. More importantly, the lack of solid findings in molecular studies demands a new look at the co-familiarity of TD and communication disturbances.

## **2.6 The environment and thought disorder**

Research on the social and environmental predictors of TD has been very sparse and has generally neglected the issue of symptom-specificity by not accounting for comorbidity psychotic experiences (e.g. hallucinations and delusions). Nevertheless, two important areas of research have produced interesting findings. One is related to

the impact of trauma and adversity on TD and the other is the historical literature on CD. In the next two sections we will address both of these risk factors separately.

### **2.6.1 Trauma and adversity**

The focus on the neurobiology of TD has led to a neglect of the role of environment in the aetiology and developmental course of TD. In fact, only a few studies have been carried out to date looking at social predictors of thought and communication disturbances.

One of these exceptions is a 10-year prospective cohort study of offspring of mothers diagnosed with schizophrenia-spectrum disorders carried out by Walker and colleagues (1981). In this cohort study information about parental absence in the first 10 years of life was gathered from the families of the high-risk children. 10 years later all the offspring were assessed for psychotic symptoms using standardised questionnaires. Interestingly, amongst all psychotic symptoms, TD was the one that showed the strongest association with history of being raised in institutionalized care ( $p < .01$ ); however, hallucinations and delusions were also found to be significantly associated with parental absence.

The second source of information on the role of the environment and adversity on TD comes from an inpatient survey carried out by Read and Argyle (1999). Both authors looked at the frequency of several psychotic symptoms in inpatients with history of sexual or physical abuse. They reported that from all the inpatients with history of sexual abuse 35% were described as displaying TD during admission. In a second study, Read and colleagues (2003) reviewed the medical files of community patients to look at the prevalence of specific psychotic symptoms in patients with history of abuse. The authors reported that 47% of the participants who had history of



adult sexual abuse had also documented evidence of TD in the case notes.

Furthermore, 71% of the patients with documented history of adult and childhood sexual abuse had also documented evidence of TD. The combination of the two types of abuse was found to be highly predictive of TD. In the discussion of the findings, Read and colleagues suggested that the diathesis for the heightened reactivity to stress (often associated with a genetic liability to schizophrenia) could in fact represent the long-lasting neurodevelopmental consequences of the abuse (Read, et al., 2001; Read, Fosse, Moskowitz, & Perry, 2014). Interestingly, this hypothesis came to be supported by later studies in the field.

Toth and colleagues (2011) carried out what is possibly the most rigorous study to date on the association of history of abuse and neglect and TD. The sample included children (mean age of 10) with history of sexual and physical abuse and neglect and controls. In order to elicit speech samples, all children were asked first to re-tell in their own words two audio-recorded stories and second asked to create new stories. All the speech samples were coded with the K-FTD (Caplan, et al., 1989). In line with previous studies, children with history of abuse and neglect exhibited significantly more illogical thinking than comparisons. In fact, the scores on the K-FTD, for the children with history of abuse and neglect, were found to be within clinical range. When the results were analysed by trauma subtype, the associations were significant for physical and sexual abuse. Toth and colleagues also found a trend towards chronicity of abuse (children abused over several developmental periods) and TD. The authors concluded:

“The dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis found in maltreated children also may lead to alterations in dopaminergic and

serotonergic system function. Such changes may help to explain why children with bipolar disorder who have been abused are more likely to experience psychotic symptoms than are similarly diagnosed children who have not been abused. Aberrant patterns of neural activity during semantic and syntactic processing tasks and structural differences in the anterior cingulate gyrus in childhood onset schizophrenia underscore possible neurodevelopmental contributors to thought disorder.” (Toth, et al., 2011, p. 665).

It is interesting to note the similarities between this proposal and Read and colleagues (2001) *traumagenic neurodevelopmental model* of psychosis, in which trauma is conceptualized as diathesis for the development of psychotic symptoms. In another paper, Read and Gumley (2008) went a step further to suggest that attachment could mediate the relationship between trauma and psychosis. As discussed earlier in this review, Dozier and Lee (1995) reported a significant correlation between deactivating attachment strategies and TD in participants with severe psychopathology. Unfortunately, to the best of our knowledge, Dozier and Lee's paper remains as the only study to date that looked at the specific relationship between attachment styles and TD. Nevertheless, it is conceivable that high levels of attachment anxiety caused by trauma may explain some of the communicational difficulties found in thought-disordered patients.

More recently, Conway and colleagues (2013) compared the narratives to the Thematic Apperception Test (TAT; Murray, 1943) of 12 year olds with history of childhood sexual abuse and controls. The narratives were coded for TD with the TLI (Liddle, et al., 2002). Again, authors reported that children with history of childhood sexual abuse achieved significantly more instances of non-logical thinking to card #4

(the card has a male and female figures) than comparisons and a significant correlation between childhood sexual abuse and non-logical reasoning was reported.

Unfortunately, no studies to date have tested the relationship between TD and history of trauma controlling for comorbid psychotic symptoms (e.g. hallucinations and delusions). This is especially relevant given the association between childhood adversities and psychosis (Varese, et al., 2012) and its potential specificity between types of adversities and psychotic experiences (e.g. childhood sexual abuse and auditory verbal hallucinations, Bentall, et al., 2012).

### **2.6.2 Family communication**

One environmental factor that has been long associated with TD in the offspring is parental communication deviance (CD; Singer & Wynne, 1965a; Singer & Wynne, 1965b; Wynne, et al., 1977; Wynne, 1984; Wynne & Singer, 1963a, 1963b). CD refers to a form of intrafamilial communication that is vague, fragmented, and contradictory and that compromises the development and sharing of meaning between parent and offspring (Wynne, 1981).

Initially developed by family therapy researchers Lyman Wynne and Margaret Singer, CD was contextualized as a broad set of communicational and linguistic disturbances (Singer & Wynne, 1966). Exposure to this style of intrafamilial communication was hypothesized to have a pervasive impact on the offspring's developing cognition, leading to TD (Wynne, 1984).

Since its initial conceptualization, the association between CD and psychosis in offspring has been independently replicated in many studies (de Sousa, Varese, Sellwood, & Bentall, 2013). However, only a few studies have looked into the

specific association between CD and TD and none as adequately controlled for comorbid psychotic symptoms.

In one of their earliest studies Wynne and Singer had been able to blindly predict severity of offspring's TD from the parents' test data ( $p < .001$ ) suggesting some degree of symptom specificity (Singer & Wynne, 1965b). Since then, other studies have also found significant associations between maternal and psychotic offspring's degree of CD (Velligan, et al., 1995) or between maternal thinking disturbance and offspring's TD (Harrow & Quinlan, 1985; Johnston & Holzman, 1979; Shenton, et al., 1989; Tompson, et al., 1997).

Sass and colleagues (1984) tested parents of psychotic offspring with different degrees of TD using both TAT cards (Murray, 1943) and a descriptive task (*d-task*)<sup>8</sup>. The researchers reported that parents of highly thought-disordered offspring had significantly higher CD scores regardless of whether they were compared to parents of controls or to parents of non-thought disordered psychotic offspring. Unfortunately, Sass and colleagues' study had three significant limitations: the sample size was small, TD was not measured directly in the offspring and the analyses of the data did not consider comorbid psychotic symptoms.

In another study, Rund (1985, 1986; Rund & Blakar, 1986) tested parents using both TAT cards and a communication conflict situation (CCS) in which the family members have to jointly find their way through a map with conflicting routes. When the author analysed the data, the sharpest contrast found was between parents of paranoid and non-paranoid participants, with the latter group displaying significantly more CD. A high proportion of the participants in the non-paranoid group had been diagnosed with disorganised schizophrenia, a subtype whose

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<sup>8</sup> In this task, parents were invited to explain concepts of the American culture as if they were talking to someone from a foreign culture.

prominent characteristic is TD and behavioural disorganisation. Unfortunately, and despite utilizing a robust multi-method design, Rund did not measure TD directly so no firm conclusions about the specificity of CD for TD can be drawn from the findings.

In a seminal study, Wahlberg and colleagues (1997) looked at the interaction between genetic-risk and CD in the prediction of TD. The design of the project was especially relevant as it allowed for the disentanglement of genetic effects given that CD was measured in the adoptive family of high- and low-risk adoptees (High- and low-risk status was determined by the presence or absence (respectively) of psychotic-spectrum diagnosis in the biological mother). Interestingly, the authors reported that it was the interaction between CD in the adoptive parent and the high-risk status of the adoptee that best predicted TD. More importantly was the fact that low CD in the adoptive parents of high-risk adoptees predicted low TD, suggesting that low CD may have a protective effect on the offspring's cognition.

In a later study, Wahlberg and colleagues (2000) replicated the same findings with the same sample but now using the thought disorder index (TDI; Johnston & Holzman, 1979). In this study, adoptees were on average 21 years of age at follow-up. Again, the authors reported a significant interaction between CD in the adoptive parent and adoptee's high-risk status predicting *idiosyncratic verbalisation* score ( $OR= 1.70$ ;  $CI\ 95\%[1.05; 2.76]$ ;  $p=.03$ ).

Metsänen and colleagues (2007) tested the adoptees from this Finnish cohort with a 12-year interval using the TDI (Johnston & Holzman, 1979). The average of adoptees' age at follow-up was 33. At index assessment 40.9% adoptees with high TDI scores had adoptive parents with equally high levels of CD as opposed to 19.4% of the adoptees who had been raised by adoptive parents low on CD ( $p=.04$ ). At

follow-up, CD worked only as a predictor of TD in low-risk adoptees meaning that low-risk adoptees raised by adoptive parents with high-levels of CD were more likely to have high TDI scores at follow-up and this difference was statistically significant. Surprisingly, CD did not predict increased TDI scores in the high-risk adoptees at follow-up.

In the discussion, Metsänen and colleagues argue that the unexpected results of the high-risk adoptees at follow-up could have been the consequence of a change in the environment (e.g. leaving the adoptive family's home). In Wahlberg and colleagues (1997) study, high-risk adoptees, when raised by parents with low CD, displayed lower TD suggesting that high-risk adoptees may be more sensitive to a change of environment. It may as well be that unmeasured environmental variables may have acted as important moderating factors. For example, Horan, et al. (2006) found a significant negative correlation between TD and smaller network size ( $r = -0.36, p < .05$ ) in a sample of patients diagnosed with schizophrenia.

Finally, Roisko and colleagues (2014), using data from the same Finnish cohort (adoptees were in their thirties), reported again significant associations between adoptive parent's CD and offspring's TD ( $p = .009$ ). More importantly, CD did not predict diagnoses of psychotic-spectrum disorders suggesting some specificity that has been argued by the same authors in a recent meta-analytical review (Roisko, Wahlberg, Miettunen, & Tienari, 2014).

## **2.7 Conclusions**

A considerable amount of research has been produced on the different aspects of TD across the years. It is fairly clear that TD is not a specific symptom of schizophrenia.

In fact, TD is better understood as a transdiagnostic phenomenon that has a continuous expression across a range of diagnostic groups and populations.

Curiously, most of the research carried out to date has neglected not just the transdiagnostic nature of TD but also its multidimensionality and heterogeneity. This is especially relevant when the aim is to investigate the psychological processes and mechanisms that underlie TD. It is reasonable to assume that one single cognitive mechanism is unlikely to explain the different phenomena that the construct comprises. A low-complexity model of TD should at least include a mechanism for disorganised forms of TD (e.g. derailment and incoherence) and a mechanism for poverty of speech.

Most of the research has also failed to acknowledge the role of the patient's personal experience in TD. Several authors have suggested that the activation of TD during conversation could actually mean that the topic being discussed is of especial relevance and difficulty to the individual. This observation is not just important for research but also has obvious clinical implications. Indeed, perhaps the best-replicated finding in the field is that communication and thought disturbances tend to worsen when the individual is asked to make a personal disclosure or to talk about an emotionally challenging topic. Some authors have suggested that during these periods of heightened arousal, patient's personal worries and concerns become salient and intertwined in the context of conversation. This could be due to a degradation of the context module with the consequent temporary depletion of inhibitory control caused by the experience of negative affect and arousal. However, it is clear that these intrusive and salient associations have great personal significance to the patient.

Two psychological mechanisms of TD seem promising. The findings from studies using the internal source-monitoring paradigm have reported very consistent

findings. We have suggested that difficulties in source-monitoring self-generated cognitions may explain instances of derailment and incoherence, given that segments of speech may inadvertently be missed. Concurrently, we have also suggested that the inadvertent verbalisation of inner speech may lead to disordered communication given the condensed and agrammatical nature of inner speech.

Studies investigating deficits at the level of ToM and social cognition have also suggested some degree of association with TD (although this mechanism seems to contribute to other psychotic experiences and is associated with important confounders). It seems reasonable to assume that poor ToM would make an individual vulnerable to conversational misalignment (e.g. tangentiality, derailment or circumstantiality) and unable to meet the communicational needs of the listener as it seems to be the case in TD. However, poor ToM alone does not have the power to explain instances of incoherence.

Less promising have been the results of semantic priming studies. Overall, the evidence for a specific association with TD seems weak. Perhaps more concerning is the fact that the study of the semantic priming effect in patients diagnosed with schizophrenia carries important limitations and confounders. Beck and colleagues have offered an alternative hypothesis suggesting that TD may be associated to hyperpriming to semantic associations that carry personal significance to the patient. This hypothesis is yet to be tested but is consistent with the idiosyncratic nature of TD.

Surprisingly, no research to date has been carried testing these cognitive mechanisms in association with genetic or environmental predictors of TD. The co-familiarity of TD and communication disturbances, which has often been presented as evidence of a genetic contribution, has not been accompanied by consistent findings



from twin studies or by the identification of genetic mechanisms in molecular studies. In fact, replication studies of specific genetic loci are non-existent.

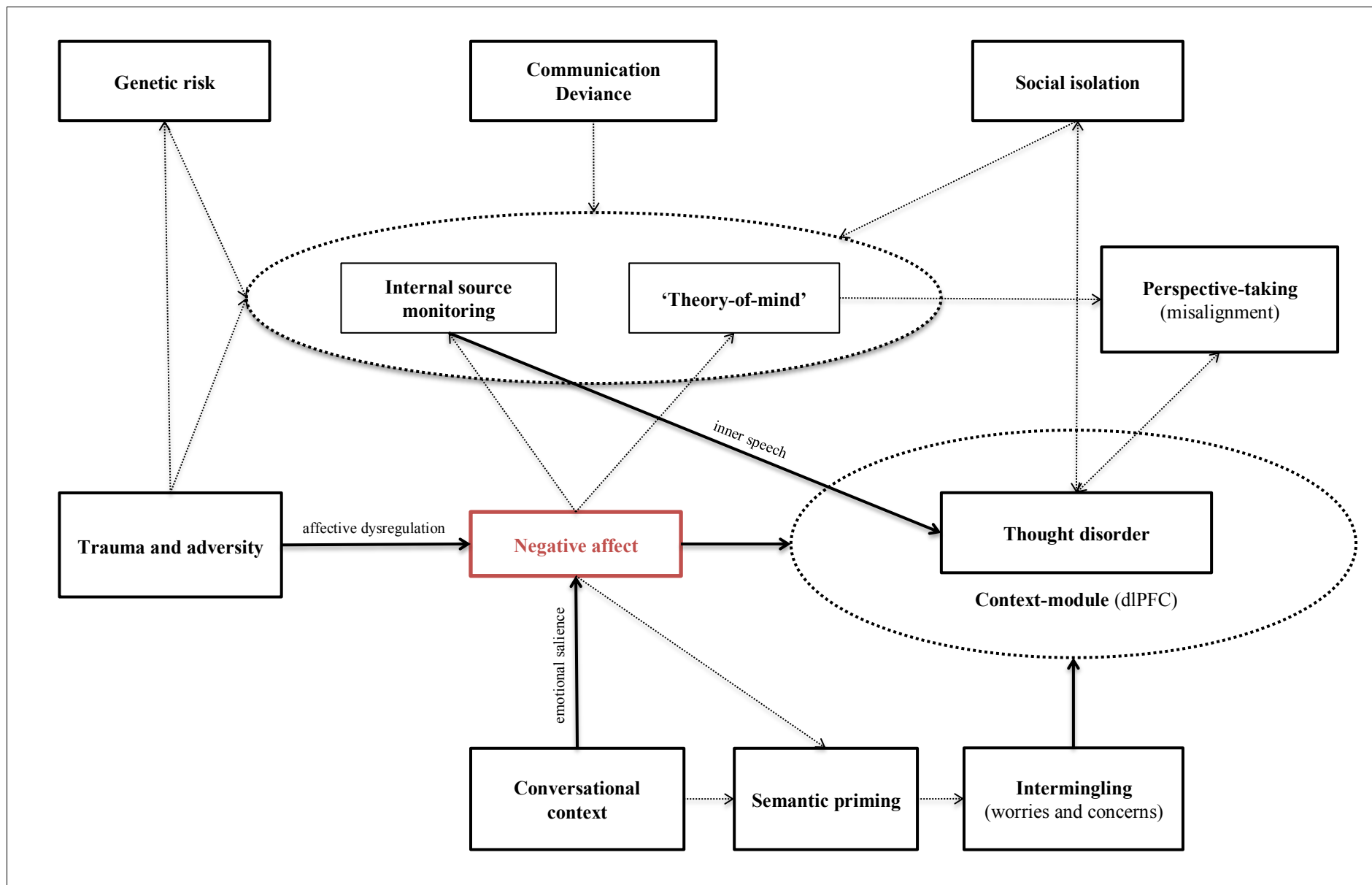
Although sparse, more promising have been the studies on the environmental and social predictors of TD. Childhood trauma, neglect and institutionalization have all been reported to be associated with TD but no study has tested more complex models involving the cognitive mechanisms of interest. Childhood adversity and trauma are not specific to TD but they are likely to make important contributions via their impact on the psychological and affective processes involved (e.g. internal source monitoring and affective dysregulation).

Family communication deviance (CD) has produced some interesting findings (e.g. gene x environment interactions predicting TD) but no study to date has controlled for symptom co-morbidity. More importantly, no study has explored potential developmental mechanisms that could explain its environmental impact. For example, family communication is likely to impact on the development of ToM and social cognition in the offspring. Obviously, we are not arguing that this is the final pathway to TD but it is a plausible contributory mechanism that deserves further investigation.

Other risk factors are likely to contribute to TD. For example, the role of the interpersonal environment has hardly received any attention. In this context, it is striking that no study to date has tested the role of social isolation in TD. Social isolation is likely to impact on the ability to source monitor self-generated cognitions (internal source monitoring) and ToM given that it deprives the individual of social interaction and communicational opportunities. Such pathway could potentially explain not just development but also maintenance of TD.

### **2.7.1 A transdiagnostic and very tentative cognitive-developmental model of TD**

In figure 3, we present a diagram of a (very) tentative transdiagnostic model of TD. This model details potential and plausible relationships between different social and environmental factors and the psychological and affective processes that have been supported by research.



**Figure 3** - A transdiagnostic and very tentative cognitive-developmental model of TD.

The model has been developed to account for instances of positive or disorganised TD and not the poverty dimension of TD (It is likely that this latter dimension may be better explained by the same processes that underlie the negative symptoms of schizophrenia).

The model offers causal relationships between environmental and cognitive variables but also developmental hypotheses that are related to the long-term impact of familial CD on psychological processes. This last point is important as most of the research on CD has been done with adult populations and using cross-sectional designs that don't clarify developmental trajectories. In the future, it would be important to study the impact of CD and its potential association with TD using longitudinal designs. The model hypothesises that the relationship between CD and TD (Roisko et al., 2014; Wahlberg et al., 2000) may be mediated by the impact of CD on the psychological mechanisms that have been found to be implicated in TD, more specifically ToM. On this issue it is important to note that family communication has been found to be an important predictor of ToM development in the offspring (Dunn, Brown, Slomkowski, Tesla, & Youngblade, 1991; Dunn, Brown, & Beardsall, 1991), which suggests that a link between CD and TD via ToM is plausible.

We also suggest that the potential association between social isolation and TD (Horan, Subotnik, Snyder, & Nuechterlein, 2006) may be explained by the impact of the former on psychological processes such as ToM and internal source monitoring.

At a more proximal level, the experience of negative affect elicited by the conversational context may lead to a temporary, and state-dependent, deterioration of psychological processes (e.g. internal source monitoring and ToM) and, at a more biological level, of the context-module (see Burbridge & Barch, 2002). For example, the deterioration of the internal source monitoring could lead to the inadvertent

verbalisation of inner speech or the omission of important segments of speech (as we mentioned before) but also to the deterioration of the ToM skills and consequentially impact on the ability to take the perspective of the listener and adapt the communication to the listener's needs (essential for conversational alignment).

In our model, the *intermingled* concerns and worries that enter the context of conversation (Harrow et al., 1983) are primed by the conversational context and enter the discourse of the thought-disordered individual due to the temporary deterioration of the context-module and consequent weakening of inhibitory processes. The model also suggests a role for trauma and adversity via its potential impact on both affective processes (affective dysregulation) and psychological processes (i.e. internal source monitoring and ToM).

Finally, a more secondary role has been reserved to genetics. This is in part due to the lack of evidence (replication) in molecular studies supporting the role of genes in TD. However, it is likely that trauma and adversity may impact on genes (e.g. through methylation) and genetic vulnerability may contribute trait-like deficits in the psychological processes involved in TD.

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**Chapter 3. The affective reactivity of psychotic speech:  
The role of internal source monitoring in explaining  
increased thought disorder under emotional challenge**

This paper has been submitted and is currently under review as de Sousa, P., Sellwood, W., Spray, A., & Bentall, R. P. (under review). The affective reactivity of psychotic speech: The role of internal source monitoring in explaining increased thought disorder under emotional challenge. *Schizophrenia Research*.

### 3.1 Abstract

**Background.** Thought disorder (TD) has been shown to vary in relation to negative affect. Here we examine the role internal source monitoring (iSM, i.e. ability to discriminate between inner speech and verbalized speech) in TD and whether changes in iSM performance are implicated in the affective reactivity effect (deterioration of TD when participants are asked to talk about emotionally-laden topics).

**Methods.** Eighty patients diagnosed with schizophrenia-spectrum disorder and thirty healthy controls received interviews that promoted personal disclosure (emotionally salient) and interviews on everyday topics (non-salient) on separate days. During the interviews, participants were tested on iSM, self-reported affect and immediate auditory recall.

**Results.** Patients had more TD, poorer ability to discriminate between inner and verbalized speech, poorer immediate auditory recall and reported more negative affect than controls. Both groups displayed more TD and negative affect in salient interviews but only patients showed poorer performance on iSM. Immediate auditory recall did not change significantly across affective conditions. In patients, the relationship between self-reported negative affect and TD was mediated by deterioration in the ability to discriminate between inner speech and speech that was directed to others and socially shared (performance on the iSM) in both interviews. Furthermore, deterioration in patients' performance on iSM across conditions significantly predicted deterioration in TD across the interviews (affective reactivity of speech).

**Discussion.** Poor iSM is significantly associated with TD. Negative affect, leading to further impaired iSM, leads to increased TD in patients with psychosis. Avenues for future research as well as clinical implications of these findings are discussed.

## 3.2 Introduction

Thought disorder (TD) is a common (Roche, Creed, MacMahon, Brennan, & Clarke, 2014) and enduring feature of psychosis (Marengo & Harrow, 1987, 1997) that is associated with poorer occupational (Racenstein, Penn, Harrow, & Schleser, 1999) and social functioning (Bowie & Harvey, 2008), poorer quality of life (Tan, Thomas, & Rossell, 2014) and relapse in patients (Wilcox, 1990), and transition to psychosis in high-risk populations (Bearden, Wu, Caplan, & Cannon, 2011). As there is a lack of evidence-based treatments for TD (Beck, Rector, Stolar, & Grant, 2009) there is a pressing need to understand the mechanisms that underlie it.

Hyperpriming in semantic memory (i.e. hyper-activation of semantically-related nodes) has been proposed as one such mechanism. However, a meta-analysis failed to find significant differences between TD and non-TD patients (Pomarol-Clotet, Oh, Laws, & McKenna, 2008). An alternative theory implicates difficulties with ‘theory-of-mind’ (ToM, Hardy-Baylé, Sarfati, & Passerieux, 2003) which could explain difficulties sharing topics and misalignment in conversation. Indeed impairments in ToM, although not specific, are highly associated with TD (Sprong, Schothorst, Vos, Hox, & van Engeland, 2007) but these difficulties alone are unlikely to explain incoherent speech.

### 3.2.1 Internal source monitoring (iSM)

iSM refers to the ability to discriminate between self-generated private stimuli such as inner speech, and self-generated speech that is directed to others (Johnson, Hashtroudi, & Lindsay, 1993) (iSM is different from external source monitoring implicated in hallucinations, in which the individual distinguishes between inner speech and the *heard* speech of others, Brookwell, Bentall, & Varese, 2013). Harvey

(1985) reported and subsequently replicated (Harvey, Earleboyer, & Levinson, 1988; Harvey & Serper, 1990) an association between TD in schizophrenia patients and a bias towards over-reporting words as having been verbalized when they had only been thought. Nienow and Docherty (2004) replicated this finding controlling for IQ and working memory and, in a later study, reported a significant association between these biases and communication disturbances (Nienow & Docherty, 2005). More recently, Docherty (2012) tested patients using both iSM and an external source-monitoring task. Again, performance on the former was found to be a significant predictor of communication disturbances even after controlling for performance on the external source monitoring, immediate recall and working memory.

### **3.2.2 Affect**

The exacerbation of communication difficulties during discussion of affectively-laden topics has been termed *affective reactivity of speech* and has been observed in schizophrenia (Docherty, 1996; Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995) and bipolar disorder (Tai, Haddock & Bentall, 2004). For example, Docherty and colleagues tested schizophrenia patients using two speech tasks in which they had to discuss stressful or pleasant experiences; participants displayed more TD in the stressful condition (Docherty, Evans, Sledge, Seibyl, & Krystal, 1994; Docherty, Sledge, & Wexler, 1994).

The goal of this study was to investigate whether increased TD during an emotionally salient interview is due to deterioration in the ability to discriminate between inner speech and speech that is directed to others (iSM). We predicted that performance on iSM as well as TD would worsen when patients were asked about emotional-laden

topics and that the worsening of iSM would be a significant predictor of the increase in TD.

### **3.3 Materials and methods**

#### **3.3.1 Participants**

Eighty participants (see Table 3) were recruited from mental health sites in the UK. The recruitment targeted 18-65 year olds with a diagnosis of schizophrenia-spectrum disorder (WHO, 2004). All participants provided informed consent according to the Declaration of Helsinki. We excluded participants whose first language was not English, who had severe learning difficulties, recent substance abuse or history of medical disorders that could affect brain function. Antipsychotic medications were converted to chlorpromazine-equivalents as per agreed conventions (Woods, 2003).

For comparison purposes, thirty healthy participants were recruited through advertisements in the community. An attempt was made to select participants who were approximately comparable for age, sex and ethnicity with participants in the clinical group (see appendix 1 for correspondence regarding ethical approval and appendices 2, 3 and 4 for consent form and information sheets).

#### **3.3.2 Materials**

##### ***3.3.2.1 Psychotic symptoms***

Psychotic symptoms were measured using the Positive and Negative Syndromes Scale (PANSS, Kay, Fiszbein, & Opler, 1987) that measures 30 symptoms, comprising a positive, a negative, and a general psychopathology scale (see appendix 5). Each item is scored from 1 to 7 with the higher score indicating increased severity. The scale has been found to have good psychometric properties (Kay et al., 1987).

### **3.3.2.2 IQ**

Intelligence was evaluated using the Quick test (QT, Ammons & Ammons, 1962) in which the participant is presented with four pictures (e.g. a policeman stopping the traffic with a whistle) and is asked to identify fifty words by pointing to the appropriate card where the word referent can be found (e.g. “whistle”; see appendix 7). The final score is achieved by summing the number of words correctly identified and scores are converted using standardised guidelines (Ammons & Ammons, 1962).

### **3.3.2.3 Interviews**

Speech samples were gathered using two interviews that had been previously developed to elicit TD (Tai, Haddock, & Bentall, 2004). The salient interview involved fifteen questions that promoted self-disclosure by asking for negative autobiographical memories, whereas the non-salient interview included fifteen questions about neutral topics (see appendix 8). Means and standard deviations for duration of the interviews and word-counts can be seen in Table 4.

### **3.3.2.4 TD**

Speech samples were rated by two independent raters, one of whom was blind to the study hypotheses, using the 18-items of the Scale for the Assessment of Thought, Language and Communication (TLC, Andreasen, 1986; see appendix 9). The total is achieved by summing the items scores. The scale has good psychometric properties (Andreasen, 1979, 1986). Table 4 shows the means and standard deviations for the total scores.

### 3.3.2.5 *Affect*

Affect was measured with the positive and negative affect scale (PANAS, Watson, Clark, & Tellegen, 1988; see appendix 11) which assesses positive and negative mood using 20 words (e.g. excited, jittery, nervous) rated by participants according to how they felt during the interview using a five-point scale. The measure has good psychometric qualities (Watson et al., 1988). Means and standard deviations for both groups across interviews can be seen in Table 4.

### 3.3.2.6 *iSM*

iSM was measured using a task developed by Nienow and Docherty (2004, 2005). Sixteen cards with a statement and a self-evident missing word are presented sequentially (e.g. “The opposite to left is \_\_\_\_\_.”). A card with the word “answer” follows half of the statement-cards. Participants are instructed to say out loud the missing word when they are presented with the “answer”-card or to just think about the missing word when the card is not presented. After the task, participants complete a recognition sheet with 24 items (8 are new words) and asked to identify the words that have been *said*, *thought* or that are *new*. The task has two versions and the order of the “answer”-cards is reversed across these (see appendix 10).

Several scores are derived from the task a) *recognition score* - total of words correctly recalled, b) *discrimination index* - words correctly recalled as either said or thought divided old words correctly recalled, c) *think-report-say errors* - words that were thought and reported as said divided by the number of old words, and d) *say-report-think errors* - words that were said but were reported as thought divided by the number of old words.

### ***3.3.2.7 Immediate auditory recall***

Immediate auditory recall was measured with the digit-span test (DST). During the task, a voice reads out a sequence of random numbers (e.g. 3, 7, 9). Immediately afterwards, the participant has to type the sequence using a keypad. We used a forward (digits must be entered by the order presented) and a backward block (digits are entered in reverse order). Each block consisted of fifteen trials plus practice trials.

### **3.3.3 Procedure**

All participants were seen on two different days to minimise ‘carry-over’ effects. Most sessions took place at the participants’ homes and the interval between them was never more than one week. Participants in the clinical group were interviewed with PANSS and comparisons screened with the Psychosis Screening Questionnaire (PSQ, Bebbington & Nayani, 1995; see appendix 6) and both were tested with the QT. Participants were then interviewed with the salient and non-salient interviews in a randomly counter-balanced order across the two sessions. Each interview followed a scripted protocol and lasted approximately 15-minutes.

All interviews were gently interrupted midway for the iSM to be completed. Each participant completed the two versions of the task across the two sessions in a counter-balanced order and the two versions were randomly assigned. After the interviews, participants were asked to score the PANAS and complete the DST. The speech samples were recorded with a digital recorder, transcribed and later coded with the TLC.



		Patients	Comparisons	
Sample size		80	30	
Sex (%)	Male	58 (72.5%)	21 (70%)	$\chi^2 = .067; p = .795$
	Female	22 (27.5%)	9 (30%)	
Ethnicity (%)	White British	74 (92.5%)	28 (93.3%)	$\chi^2 = .006; p = .936$
	Other	6 (7.5%)	2 (6.6%)	
Age (years)		39.3 (11.6)	38.4 (13.3)	$t = .33; p = .746$
Years of education		11.2 (1.9)	12.7 (2.3)	$t = -3.35; p = .001$
IQ		98.4 (10.6)	109.5 (8.3)	$t = -5.18; p < .005$
Diagnoses (%)	Schizophrenia (F20)	48 (60%)	N/a	
	Schizoaffective (F25)	18 (22.5%)	N/a	
	Other Psychoses (F29)	14 (17.5%)	N/a	
Duration of illness (years)		15.2 (10.9)	N/a	
History of admission (yes)		73 (91.3%)	N/a	
FGA (%)		26 (23.6%)	0 (0%)	
SGA (%)		58 (72.5%)	0 (0%)	

'Mood stabilizers' (%)		14 (17.5%)	0 (0%)
Anti-depressants (%)		31 (38.7%)	0 (0%)
Equivalent CPZ dose (mg)		469.7 (389.1)	N/a
PANSS	Positive	17.1 (5.2)	N/a
	Negative	14 (4.7)	N/a
	General	38.6 (9.2)	N/a
	Total	69.8 (16.1)	N/a

**Table 3** - Clinical and demographic variables.

### **3.3.4 Data analysis**

Statistical analyses were carried out on SPSS (IBM, 2012). *t*-tests and  $\chi^2$  were used to characterize and compare the groups on demographic, cognitive and affective variables. ANOVAs were used to compare different variables between groups and across conditions. We used bivariate correlations and linear regressions to explore relationships between the different variables and hierarchical regressions to test if performance on iSM mediated the relationship between negative affect (NA) and TD in both conditions separately and with change scores.

## **3.4 Results**

### **3.4.1 Demographic and clinical variables**

Table 3 shows demographic, cognitive and clinical measures. The groups did not differ for sex, age or ethnicity. Our comparison group had more years of education and higher scores on the QT.

### **3.4.2 Key study variables**

PS and AS independently coded 10% (22) of the speech samples for reliability (AS was blind to the study's hypotheses). Kappa values were substantial with tangentiality achieving the highest level of agreement ( $K = .82$ ) and self-reference the lowest ( $K = .62$ ). Because some items of the TLC are dependent on word-count (e.g. poverty of speech), we did not adjust TD for verbosity.

As there was a) no association between positive affect and TD (see Table 5) and b) previous research has indicated that positive affect is not related to TD (Cohen & Docherty, 2005), only the negative scale was analysed.

	Neutral condition				Salient condition			
	Patients		Comparisons		Patients		Comparisons	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
TD	8.16	6.14	1.77	2.02	12.35	9.31	3.93	2.79
Duration (mins: secs)	15:03	01:34	15:03	00:22	15:15	02:13	15:02	00:25
Word count	1388.04	556.26	2046.37	315.14	1424.45	622.57	2042.93	359.13
Recognition score <sup>1</sup>	.60	.14	.74	.10	.50	.17	.72	.10
Discrimination index <sup>2</sup>	.68	.14	.77	.13	.57	.19	.76	.12
Say-report-think errors <sup>3</sup>	.27	.19	.30	.19	.33	.21	.29	.19
Think-report-say errors <sup>4</sup>	.26	.21	.10	.10	.38	.28	.13	.13
DST forward	6.25	1.45	7.33	.99	6.25	1.33	7.4	.77
DST backward	4.86	1.52	6.2	.84	4.71	1.49	6	1.23
Positive affect <sup>5</sup>	32.5	7.7	34.6	6.4	27.7	8	29.4	7.8
Negative affect <sup>5</sup>	14.5	5.2	11.1	1.6	23.6	7.6	16.4	4.8

<sup>1</sup>correctly identified words as old and new divided by 24. <sup>2</sup>thought and said words correctly identified divided by the total amount of old words correctly identified. <sup>3</sup>said words that were reported as being thought divided by 8. <sup>4</sup>thought words that were reported as being said divided by 8. <sup>5</sup>scores range from 0 to 50.

**Table 4** - Means and standard deviations for TD, iSM scores, DST and reported affect across group and interview.

When we ran a 2x2 ANOVA using NA scores as the dependent variable, the main effect for interview,  $F[1,108]= 122.1, p< .001, \eta_p^2 = .531$ , and the group effect were significant,  $F[1,108]= 24.44, p< .001, \eta_p^2 = .185$ , with the clinical group reporting more NA than comparisons. The interaction was also significant,  $F[1,108]= 8.56, p=.004, \eta_p^2 = .073$ , as the increase in NA across interviews was greater in the clinical group.

A 2x2 ANOVA on the TD scores revealed a main effect for interview,  $F[1,108]= 38.33, p< .001, \eta_p^2 = .262$ , and for group,  $F[1,108]= 28.93, p< .001, \eta_p^2 = .211$ , but the interaction was not significant,  $F[1,108]= 3.88, p=.052, \eta_p^2 = .035$ . Both patients and comparisons demonstrated affective reactivity of speech respectively,  $t(79)= -6.91, p< .001$  and  $t(29)= -4.99, p< .001$ .

When we compared performance across groups and interviews, using the iSM discrimination index as the dependent variable (as recommended in the literature, Docherty, 2012; Harvey, 1985) the main effects for condition,  $F[1,108]= 13.36, p< .001, \eta_p^2 = .110$  and group were significant,  $F[1,108]= 22.43, p< .001, \eta_p^2 = .172$ , as was the interaction,  $F[1,108]= 8.74, p=.004, \eta_p^2 = .075$ , with the clinical group showing a greater deterioration in the salient interview. Patients but not comparisons had a poorer discrimination index in the salient interview,  $t(79)= 5.86, p< .001$  and,  $t(29)= .556, p=.582$ , respectively.

In the case of forward DST, the group effect was significant,  $F[1,108]= 19.53, p< .001, \eta_p^2 = .153$ , but the effect for condition,  $F[1,108]= .107, p=.744, \eta_p^2 = .001$  and the interaction were not,  $F[1,108]= .107, p=.744, \eta_p^2 = .001$ . With DST backwards as the dependent variable, there was also an effect for group,  $F[1,108]=$

21.3,  $p < .001$ ,  $\eta_p^2 = .165$  but again not for condition,  $F[1,108] = 3.32$ ,  $p = .071$ ,  $\eta_p^2 = .03$  or for the interaction,  $F[1,108] = .068$ ,  $p = .795$ ,  $\eta_p^2 = .001$ .

### 3.4.3 iSM and affective reactivity in patients

Table 5 shows bivariate correlations for the patients between affect, TD and iSM indices in the two conditions. In the neutral interview, TD scores correlated with two of the iSM measures (the discrimination index and think-report-say errors) and with NA. In the salient interview, TD correlated with all iSM scores and again with NA.

We ran hierarchical linear regressions on TD scores for both conditions separately, with NA and order of presentation of the conditions entered first and then discrimination scores entered in a second stage. For the neutral condition, NA predicted TD,  $F[2,77] = 4.47$ ,  $p = .015$ ,  $\beta = .32$ ,  $p = .004$ . Adding discrimination scores led to an improved model,  $F[3,76] = 14.20$ ,  $p < .001$ , with the effect for NA no longer significant,  $\beta = .16$ ,  $p = .101$ , but with the discrimination index as a significant predictor,  $\beta = -.54$ ,  $p < .001$ .

For the salient condition, the initial model was again significant,  $F[2,77] = 5.53$ ,  $p = .006$ , with NA predicting TD,  $\beta = .35$ ,  $p = .002$ . Adding the discrimination index improved the model,  $F[3,76] = 16.05$ ,  $p < .001$ ; the significance of NA was reduced,  $\beta = .22$ ,  $p = .024$  and the discrimination index was a significant predictor of TD,  $\beta = -.53$ ,  $p < .001$ .

Neutral	1.	2.	3.	4.	5.	6.	7.	1.	2.	3.	4.	5.	6.	7.
1. TD	-													
2. Recognition score	<b>-.684<sup>***</sup></b>	-												
3. Discrimination index	<b>-.561<sup>***</sup></b>	<b>.805<sup>***</sup></b>	-											
4. Say-report-think errors	.202	<b>-.353<sup>***</sup></b>	<b>-.370<sup>***</sup></b>	-										
5. Think-report-say errors	<b>.465<sup>***</sup></b>	<b>-.497<sup>***</sup></b>	<b>-.364<sup>***</sup></b>	-.212	-									
6. Positive affect	-.029	.081	.019	.098	-.143	-								
7. Negative affect	<b>.314<sup>**</sup></b>	<b>-.299<sup>**</sup></b>	<b>-.300<sup>**</sup></b>	.023	<b>.258<sup>*</sup></b>	-.214	-							
Salient														
1. TD	<b>.831<sup>***</sup></b>	<b>-.544<sup>***</sup></b>	<b>-.435<sup>***</sup></b>	.183	<b>.403<sup>***</sup></b>	.036	<b>.291<sup>**</sup></b>	-						
2. Recognition score	<b>-.718<sup>***</sup></b>	<b>.697<sup>***</sup></b>	<b>.590<sup>***</sup></b>	-.126	<b>-.471<sup>***</sup></b>	-.046	<b>-.290<sup>**</sup></b>	<b>-.709<sup>***</sup></b>	-					
3. Discrimination index	<b>-.523<sup>***</sup></b>	<b>.482<sup>***</sup></b>	<b>.457<sup>***</sup></b>	-.147	<b>-.359<sup>***</sup></b>	-.132	-.205	<b>-.583<sup>***</sup></b>	<b>.816<sup>***</sup></b>	-				
4. Say-report-think errors	<b>.321<sup>**</sup></b>	-.194	<b>-.241<sup>*</sup></b>	.183	.099	.215	.044	<b>.303<sup>**</sup></b>	<b>-.369<sup>***</sup></b>	<b>-.303<sup>**</sup></b>	-			
5. Think-report-say errors	<b>.452<sup>***</sup></b>	<b>-.622<sup>***</sup></b>	<b>-.361<sup>***</sup></b>	.062	<b>.465<sup>***</sup></b>	-.024	.125	<b>.429<sup>***</sup></b>	<b>-.658<sup>***</sup></b>	<b>-.604<sup>***</sup></b>	-.021	-		
6. Positive affect	-.041	.120	.051	.005	-.120	<b>.597<sup>***</sup></b>	-.114	.024	.027	-.135	-.042	.014	-	
7. Negative affect	<b>.290<sup>**</sup></b>	-.199	-.142	-.112	.174	.062	<b>.516<sup>***</sup></b>	<b>.352<sup>***</sup></b>	<b>-.359<sup>***</sup></b>	<b>-.244<sup>*</sup></b>	<b>.298<sup>**</sup></b>	.097	.022	-

Note: \*  $p < .05$  \*\*  $p < .01$  \*\*\*  $p < .001$

**Table 5** - Bivariate correlations between TD, iSM and affective scores for the clinical group.

Finally, in order to test whether *change* in discrimination indices across interviews was a significant predictor of affective reactivity of speech, we calculated difference scores for NA, the discrimination index and TD by subtracting scores from the neutral from those of the salient condition. In a hierarchical linear regression with affective reactivity of speech as the dependent variable, we entered order of presentation, adding affect change in the second step and then the change in discrimination index in the third (see Table 6). In this analysis, the initial association between the increase in NA and the increase in TD was not significant. However, the change in iSM was a significant predictor of the increase in TD when it was added to the model.

### 3.5 Discussion

Replicating previous findings, we found that psychotic patients display more TD when discussing emotionally salient topics and, consistent with results from Tai and colleagues (2004), we also found the less marked affective reactivity of speech in healthy comparisons, suggesting that it occurs on a continuum with healthy functioning. Secondly, and consistent with previous studies, we found that patients performed considerably worse on the iSM task (Harvey, 1985). In both conditions, nearly all of the scores on this task (with the exception of *say-report-think errors* in the neutral interview) were substantially associated with TD.

The novel aspect of this research concerned the role of the emotional and cognitive variables in the affective reactivity effect. Our clinical group reported more NA during the interviews (especially the salient interview), which is consistent with the literature on emotional experience in schizophrenia (Cohen & Minor, 2010). Although performance on the DST was not affected by condition, the discrimination indices on the iSM were, but the effect



	B	Standard error	Beta	<i>t</i>	<i>p</i> -value	95% CI	
1. Dependent variable: $\Delta$ Thought disorder							
Order of condition	1.166	1.212	.108	.962	.339	-1.247	3.580
2. Dependent variable: $\Delta$ Thought disorder							
Order of condition	.672	1.235	.062	.544	.588	-1.787	3.132
$\Delta$ Negative affect	.155	.093	.190	1.661	.101	-.031	.340
3. Dependent variable: $\Delta$ Thought disorder							
Order of condition	.224	1.202	.021	.187	.852	-2.170	2.619
$\Delta$ Negative affect	.119	.091	.147	1.313	.193	-.062	.300
$\Delta$ Discrimination index	-.609	.231	-.290	-2.635	.010	-1.069	-.149

**Table 6** - Analysis of mediation, negative affect (x) on thought disorder (y) through discrimination index (m) for the clinical group.

was only observed in patients. This observation provided preliminary evidence that iSM was implicated in the increased TD seen in the emotionally salient condition.

Our regression analyses showed that, in the patients and in each interview, the relationship between NA and TD was mediated by deterioration in iSM. However, our final analysis based on change scores was less clear-cut. On the one hand, the observed decrements in iSM predicted the increase in TD as expected, supporting the meditational hypothesis. However, the expected association between increased NA and increased TD was not significant. Although this observation might be thought to cast doubt about the chain of processes from negative emotion through impaired source monitoring to TD, it is worth noting that the use of change scores may have introduced additional noise into the data set. Another possibility is that our measure of affect did not sufficiently pick out the specific emotional response that leads to increased TD. Overall, given the evidence that iSM mediated between NA and TD in each condition and that impairment in iSM predicted the increase in TD, we tentatively conclude that the data supports the hypothesis that impaired iSM plays a role in the affective reactivity effect.

Consistent with previous findings (Nienow & Docherty, 2004, 2005) think-report-say errors were significantly associated with TD whereas say-report-think errors were significantly associated with TD only in the salient interview. A difficulty discriminating between inner speech and speech that is socially directed is likely to compromise communication by either leading to the omission of segments of speech or by the inadvertent verbalisation of inner speech. The former phenomenon would deprive the listener of crucial information for shared understanding. The latter would, for the listener, involve listening to the patient's stream-of-consciousness, in which

case the jumbled up quality of TD could be construed as the condensed nature of inner speech (Vygotsky, 1987).

There are several limitations to this study. The patients and controls were not matched on education or IQ. Also, our interview protocol was different from an everyday conversation. It would be interesting to use more naturalistic speech samples e.g. everyday family conversations; which could facilitate investigation of the impact on TD of those aspects of family communication that have been previously reported to be important in TD (de Sousa, Varese, Sellwood, & Bentall, 2013).

In future research it could be informative to assess increases in cortisol secretion as well as explore the role of other variables such as ToM which have been implicated in conversational alignment (Pickering & Garrod, 2006). Finally, therapeutic techniques such as role-playing, five-sentence rule, or relaxation breaks have been suggested to address TD (Beck et al., 2009). An alternative approach would be to develop interventions that specifically target the mechanisms identified in this study. In the case of NA, we subscribe to the suggestion that the therapeutic focus should be on emotion regulation techniques (Beck et al., 2009). Future research should consider the utility of cognitive rehabilitation techniques to improve iSM.

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## **Chapter 4. Inner speech and clarity of self-concept in thought disorder and auditory-verbal hallucinations**

This paper has been submitted and is currently under review as de Sousa, P., Sellwood, W., Spray, A., Fernyhough, C., & Bentall, R. P. (under review). Inner speech and clarity of self-concept in thought disorder and auditory-verbal hallucinations. *Journal of Nervous and Mental Disease*.

## 4.1 Abstract

**Background.** This study investigated relationships between clarity of the self-concept, quality of inner speech and psychotic symptoms in patients diagnosed with ICD-10 schizophrenia-spectrum disorders.

**Methods.** Eighty patients and thirty controls were interviewed twice using one interview that promoted personal disclosure and another that invited talk about everyday topics. Speech was scored for thought disorder (TD) with the Thought, Language and Communication scale (TLC). All participants completed the Self-Concept Clarity Scale (SCCS) and the Varieties of Inner Speech Questionnaire (VISQ). Co-morbid psychotic experiences were measured with the Positive and Negative Syndromes Scale (PANSS).

**Results.** Patients scored lower than comparisons on the SCCS. Low scores were associated with TD, and, more specifically, the disorganised dimension of TD ( $r = -0.265, p < .05$ ). Patients also scored significantly higher on condensed inner speech and other people in inner speech subscales of the VISQ, but not on dialogical or evaluative inner speech. The poverty of speech dimension of TD was associated with less dialogical inner speech ( $r = -0.457, p < .001$ ), less other people in inner speech ( $r = -0.282, p < .05$ ) and less evaluative inner speech ( $r = -0.323, p < .01$ ). Hallucinations were significantly associated with more other people in inner speech ( $r = 0.534, p < .001$ ) and more evaluative inner speech ( $r = 0.264, p < .05$ ).

**Conclusion.** Clarity of self-concept and qualities of inner speech are differentially associated with individual TD dimensions. The findings also support inner speech models of auditory verbal hallucinations.

“Truth is not born nor is it to be found inside the head of an individual person; it is born between people collectively searching for truth, in the process of their dialogic interaction.”(Bakhtin, 1929, p.110).

## **4.2 Introduction**

Thought Disorder (TD) refers to a heterogeneous and transdiagnostic cluster of cognitive, linguistic, and communication disturbances that compromise the sharing of meaning during conversation (Andreasen, 1986; Cuesta & Peralta, 1999) and that are highly prevalent in schizophrenia with some estimates reaching 91% (Roche, Creed, MacMahon, Brennan, & Clarke, 2014). TD has been found to be significant predictor of conversion into psychosis in high-risk populations (Bearden, Wu, Caplan, & Cannon, 2011; Cannon et al., 2008; Ott, Roberts, Rock, Allen, & Erlenmeyer-Kimling, 2002) and has been associated with a range of adverse outcomes such as psychotic relapse (Wilcox, 1990), poorer occupational (Racenstein, Penn, Harrow, & Schleser, 1999) and social functioning (Bowie, Gupta, & Holshausen, 2011; Bowie & Harvey, 2008), and poorer quality of life (Tan, Thomas, & Rossell, 2014). Despite its clinical relevance, TD is still a poorly understood phenomenon and evidence-based therapeutic approaches are nearly non-existent (Beck, Rector, Stolar, & Grant, 2009).

A variety of theories have been produced to explain TD, from poor executive ability (Kerns & Berenbaum, 2002; McGrath, 1991; McGrath, Scheldt, Hengstberger, & Dark, 1997; Stirling, Hellewell, Blakey, & Deakin, 2006), disorganisation of semantic networks (Goldberg & Weinberger, 2000; Goldberg et al., 1998), a hyperpriming effect in semantic memory (Pomarol-Clotet, Oh, Laws, & McKenna, 2008; Spitzer, 1997) to deficits at the level of context representation (Cohen &

Servan-Schreiber, 1992; Roesch-Ely, Spitzer, Kaiser, Weisbrod, & Pfueller, 2010). Neurobiological correlates include decreased grey matter volume in the left posterior superior temporal gyrus which has also been associated with auditory verbal hallucinations (Shenton et al., 1992; Subotnik, Bartzokis, Green, & Nuechterlein, 2003; Vita et al., 1995), decreased activity in the inferior frontal, cingulate and left superior temporal cortex whilst patients are asked to describe ambiguous pictures (McGuire et al., 1998) and abnormal dorsolateral prefrontal activity during functional magnetic resonance imaging studies (Goghari, Sponheim, & MacDonald, 2010; Roesch-Ely et al., 2010).

It has been argued that the perceived unintelligibility of TD (Beck et al., 2009; Bentall, 2003) may in fact reflect the intermingling of decontextualized personal concerns and worries (Harrow, Lanin-Kettering, Prosen, & Miller, 1983; Lanin-Kettering & Harrow, 1985) coupled with a loss of perspective (Harrow et al., 2000) or poor theory of mind (Frith, 1992; Hardy-Baylé, Sarfati, & Passerieux, 2003) making it difficult for the speaker to adjust their speech according to the needs of the listener. TD has been observed to become more pronounced when patients are asked to disclose negative autobiographical memories (Shimkunas, 1972; Tai, Haddock, & Bentall, 2004) or affect-laden material (Docherty, Evans, Sledge, Seibyl, & Krystal, 1994; Docherty, Hall, & Gordinier, 1998; Docherty, Sledge, & Wexler, 1994; Docherty, 1996; Mohagheghi, Farnam, Farhang, & Bakhshipoor, 2012).

#### **4.2.1 TD as disruption of inner dialogue**

One outstanding question concerns whether the organisation of the self-construct and the corresponding production of a self-narrative, impacts upon patients' ability to engage in patterned and organised dialogues with others.

A useful theoretical framework within which it is possible to consider this question is Dialogical Self Theory (DST, Hermans, Kempen, & Van Loon, 1992), which draws on philosophy (James, 1983; Nietzsche, 1997) and literary scholarship (Bakhtin, 1929) in understanding the self as an assembly or *society* of coexisting internal and external self-positions (or *I*-positions), which are hierarchically arranged, and in which the self is the dialogical narrator (Hermans et al., 1992; Paul H. Lysaker & Lysaker, 2010). Internal self-positions refer to our different representations of our identity and social roles (e.g., *I*-as a husband or *I*-as a jazz lover) whereas external self-positions are the people that populate our worlds and to whom we are affectively bonded (e.g., my friend who also loves jazz). A coherent sense of self is dependent on the communication or dialogue between the different self-positions that can be either complementary or contradictory. Internal coherence is achieved and sustained through the dynamic generated by this inner dialogue and by outer dialogue with others.

It has been argued that the disturbances of self-experience documented in psychosis, such as diminished sense of identity and agency (Frith, 1992; Sass, 2014), are related to a collapse of the dialogue of self-positions within the individual and between the individual and other people (Lysaker & Lysaker, 2005; Lysaker & Lysaker, 2002; Lysaker & Lysaker, 2001). Lysaker and Lysaker (2002) have proposed three types of potential disruptions to dialogue: (1) a suspension of inner and outer dialogue e.g. poverty of speech; (2) lack of a dialogical hierarchy and socially-validated coherence e.g. positive TD (Lysaker & Lysaker, 2006); and (3) the compromise of dialogue rigid self-position e.g. delusional beliefs.

Some studies have examined the self-concept in patients diagnosed with schizophrenia. For example, Cicero and colleagues (Cicero, Becker, Martin, Docherty, & Kerns, 2012) reported that the interaction between poor self-concept

clarity and aberrant salience was a significant predictor of psychotic-like experiences whereas a larger body of research has suggested a more specific association between negative self-concept and paranoia (Tiernan, Tracey, & Shannon, 2014). According to DST, however, TD should relate to a lack of self-concept clarity resulting from the emergence of a cacophonous self.

It should also be possible to trace disruptions to the inner dialogue through the phenomenon of inner speech, the internal flow of verbal thought that characterizes many people's conscious experience (Fernyhough, 2013). According to the Dialogic Thinking Model (DTM, Fernyhough, 1996, 2009), inner speech has a dialogic character, which reflects its developmental origins in social exchanges (Vygotsky, 1934). Inner speech has also been proposed to exist in different forms corresponding to different levels of expansion and dialogicity (Fernyhough, 2004), a proposal that has received empirical support (Alderson-Day et al., 2014; McCarthy-Jones & Fernyhough, 2011). Fernyhough (2004) proposed that auditory verbal hallucinations (AVHs) in schizophrenia might be caused by a disruption to the process whereby condensed inner dialogue is expanded into a more overt internal dialogue between differing points of view. However, this hypothesis was not supported in a study by Langdon and colleagues (2009), which showed a non-significant trend towards reduced dialogicity in inner speech in schizophrenia patients with AVHs but no significant differences in inner speech quality compared with healthy controls.

#### **4.2.2 Aims of the study**

In the present study, we wanted to investigate if lack of self-concept clarity was more prevalent in patients diagnosed with schizophrenia and, if so, whether this lack of clarity was significantly associated with TD during an interview designed to promote



personal disclosure. In line with predictions from both DST and the DTM, we also wanted to test whether patients diagnosed with schizophrenia reported experiencing less dialogic inner speech and if this might be associated with TD. At a more exploratory level we wanted to test how these variables related to the different dimensions of TD.

A secondary purpose of the present study was to also test hypotheses about the relationship between inner speech and hallucinations. Given that previous studies with non-clinical samples have found associations between both other people in inner speech and motivational/ evaluative inner speech and proneness to auditory hallucinations (Alderson-Day et al., 2014) whereas clinical studies have not (Langdon et al., 2009).

## **4.3 Methods**

### **4.3.1 Participants**

As part of a wider study of the determinants of TD, we recruited 80 clinical participants (see Table 7) from local mental health sites across the North West of England. The recruitment targeted 18–65 year olds with a psychotic-spectrum disorder as primary diagnosis defined as schizophrenia, schizoaffective, or unspecified non-organic psychosis according to ICD-10 (World Health Organization, 2004).

We excluded participants who lacked capacity for informed consent, whose first language was not English, and individuals with diagnosed learning difficulties, recent substance abuse or a history of neurological disorders. Antipsychotic medications were converted to chlorpromazine equivalents as per agreed conventions (Woods, 2003). For comparison purposes, 30 participants were recruited through local

advertisements in the community and screened for psychotic symptoms with the Psychosis Screening Questionnaire (PSQ, Bebbington & Nayani, 1995; see appendix 6). An attempt was made to select participants who were approximately comparable with our participants in the clinical group on variables such as sex, age, and ethnicity (see appendix 1 for correspondence regarding ethical approval and appendices 2, 3 and 4 for consent form and information sheets).

### **4.3.2 Measures**

#### **4.3.2.1 Psychotic symptoms**

Psychotic symptoms were measured using the *Positive and Negative Syndromes Scale* (PANSS, Kay, Fiszbein, & Opler, 1987; see appendix 5). The PANSS is a widely used clinical interview that measures 30 symptoms, comprising a positive symptom subscale, a negative symptom subscale, and a general psychopathology subscale. Each item is scored from 1 to 7 with the higher score indicating increased severity. The scale has been found to have good psychometric properties (Kay et al., 1987).

#### **4.3.2.2 Quick Test**

Verbal intelligence was measured using the Ammon's Quick test (QT, Ammons & Ammons, 1962), an untimed picture vocabulary test. The participant is presented with four pictures of different situations and is asked to identify fifty progressively difficult words by simply pointing to the appropriate card where the word referent can be found and the number of words correctly identified yields the total score (see appendix 7). The QT has been extensively used in clinical studies and correlates with WAIS scores (Lezak, 2004).

#### **4.3.2.3 Interviews**

Speech samples were gathered from all participants using two interview protocols previously developed to elicit TD (Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995; Tai et al., 2004). The protocols elicited speech samples relating to emotionally-laden (salient interview) and neutral (non-salient interview) topics, given the evidence that participants diagnosed with psychosis show more TD when asked to talk about emotional material (Docherty, Evans, et al., 1994; Docherty, 2005; Shimkunas, 1972). The salient interview involved fifteen questions that promoted self-disclosure by asking for negative autobiographical memories, whereas the non-salient interview included fifteen questions that did not promote self-disclosure (see appendix 8 for interview items).

#### **4.3.2.4 TD**

The speech samples were rated using the Scale for the Assessment of Thought, Language and Communication (TLC, Andreasen, 1986), a widely used scale that provides definitions and scores for 18 different items of TD (see Table 1) and has been supported by researchers in the field (Roche et al., 2014). The different categories of TD are rated on a scale of severity ranging from 0 to 4 or 0 to 3 (see appendix 9). The global rating is achieved by summing the scores of the different subscales. The scale can be applied to any speech samples and has been shown to have good psychometric properties (Andreasen, 1979a, 1986).

#### **4.3.2.5 Self-concept clarity**

The Self-Concept Clarity Scale (SCCS, Campbell et al., 1996; see appendix 14) is a self-report questionnaire of 12 items which measures the extent to which beliefs about

self are clearly defined, stable, and consistent. All the items are presented as sentences (e.g., “In general, I have a clear sense of who I am and what I am.”) and the participant has to choose on a scale of 5 (1 = strongly disagree to 5 = strongly agree) how that statement reflect their own perception about their self. Psychometric properties of the scale have been found to be very good (Campbell et al., 1996) and the scale has been used in psychosis research (Cicero et al., 2012). In the present sample, the Cronbach alpha coefficient was .92.

#### **4.3.2.6 Quality of Inner Speech**

The Varieties of Inner Speech Questionnaire (VISQ, McCarthy-Jones & Fernyhough, 2011; see appendix 13) is a self-report questionnaire designed to assess the phenomenological properties of inner speech. The VISQ has 18 items presented in the form of sentences (e.g., “I talk back and forward to myself in my mind about things.”), which the participant has to endorse using a six-point Likert scale (ranging from 6= “Certainly applies to me.” to 1=“Certainly does not apply to me.”). The questionnaire is composed of subscales, namely: (1) dialogic inner speech, (2) condensed inner speech, (3) other people in inner speech, and (4) evaluative and motivational inner speech. The scale has been found to have good psychometric properties (Alderson-Day et al., 2014; McCarthy-Jones & Fernyhough, 2011). Cronbach alphas for the current sample were: dialogical inner speech,  $\alpha = .85$ ; condensed inner speech,  $\alpha = .67$ ; other people in inner speech,  $\alpha = .90$ ; motivational/evaluative inner speech,  $\alpha = .81$ .

### **4.3.3 Procedure**

The present study was part of a larger research project on the social, cognitive, and affective predictors of TD approved by UK National Research Ethics Service (NRES).

All participants in the study were seen twice on different days. The interval between the two sessions was in most cases a few days and never more than one week to prevent ‘carry-over’ effects. Participants in the clinical group were interviewed with the PANSS (Kay et al., 1987) whereas controls were screened with the PSQ (Bebbington & Nayani, 1995). Following these assessments, participants completed the QT prior to being interviewed.

Each participant was interviewed using the salient and non-salient interviews (Haddock et al., 1995; Tai et al., 2004) in a randomly counterbalanced order across the two sessions. Interviews lasted approximately 15 minutes on average, providing authors with 30 minutes of speech per participant. In the second session, participants were requested to complete both the VISQ and the SCC questionnaires. The speech samples were recorded with a digital voice recorder (Olympus VN711 PC 2GB) and later transcribed by the first author and a professional transcriber, before being coded independently by PS and AS using the TLC.

### **4.3.4 Statistical analysis**

Statistical analyses were carried out on IBM SPSS Statistics (21.0.0). We used  $\chi^2$ , *t*-tests and  $2 \times 2$  mixed ANOVA to compare groups on both demographic and clinical variables. To further explore relationships between variables, we conducted bivariate and partial correlations as well as two-staged linear regressions. Finally, to determine the different dimensions of TD, we conducted a factor analysis using an unweighted

least squares method with Varimax rotation. The cut-off criterion for the factors was *eigenvalues* greater than 1.

## **4.4 Results**

### **4.4.1 Demographic and clinical variables**

The descriptive statistics for the demographic and clinical variables can be found elsewhere (de Sousa, Sellwood, Spray, & Bentall, submitted). Descriptive statistics can be found in Table 7. Briefly, the groups did not differ significantly on variables such as sex, age, or ethnicity. The only significant differences were on years of education with our comparisons reporting more years of education. The means and standard deviations of the PANSS factors approximate to the values reported in other patient studies (Kay et al., 1987). Details about the TLC reliability can be also be found in the previous paper, as well as data for the TD ratings and negative affect for both groups. Briefly, Kappa values were of substantial magnitude with tangentiality achieving the highest level of agreement ( $K = .82$ ) and self-reference the lowest ( $K = .62$ ).

As detailed elsewhere (de Sousa et al., submitted), our clinical group exhibited more TD than our comparison group, especially during the salient interview. They also reported more negative affect in both interviews and, as expected, this difference was more pronounced in the salient interview.

		Patients	Comparisons	
Number		80	30	
Sex (%)	Male	58 (72.5%)	21 (70%)	$\chi^2(1) = .07$ ; $p = .485$
	Female	22 (27.5%)	9 (30%)	
Ethnicity (%)	White British	74 (92.5%)	28 (93.3%)	$\chi^2(3) = 1.16$ ; $p = .560$
	White Irish	2 (2.5%)	1 (3.3%)	
	Black British	3 (3.8%)	0 (0%)	
	Other	1 (1.3%)	1 (3.3%)	
Age (years)		39.3 (11.6)	38.4 (13.3)	$t(108) = .33$ ; $p = .746$
Education (years)		11.2 (1.9)	12.7 (2.3)	<b><math>t(108) = -3.35</math>; <math>p &lt; .001</math></b>
IQ		98.4 (10.6)	109.5 (8.3)	<b><math>t(108) = -5.18</math>; <math>p &lt; .001</math></b>
Diagnoses (%)	Schizophrenia (F20)	48 (60%)	N/a	
	Schizoaffective (F25)	18 (22.5%)	N/a	
	Other Psychoses (F29)	14 (17.5%)	N/a	
Equivalent CPZ (mg)		469.7 (389.1)	N/a	
TD	Non-salient	8.16 (6.143)	1.77 (2.019)	<b><math>t(108) = 5.58</math>; <math>p &lt; .001</math></b>
	Salient	12.35 (9.312)	3.93 (2.791)	<b><math>t(108) = 4.86</math>; <math>p &lt; .001</math></b>
PANSS	Positive	17.1 (5.2)	N/a	
	Negative	14 (4.7)	N/a	
	General	38.6 (9.2)	N/a	
	Total	69.8 (16.1)	N/a	
Self-concept		31.31 (9.44)	44.50 (10.07)	<b><math>t(108) = -6.41</math>; <math>p &lt; .001</math></b>
Inner speech	Dialogical	16.06 (5.42)	15.30 (5.63)	$t(108) = .650$ ; $p = .517$
	Condensed	16.24 (5.19)	12.40 (5.24)	<b><math>t(108) = 3.44</math>; <math>p &lt; .001</math></b>
	Other people	17.10 (7.78)	7.77 (3.23)	<b><math>t(108) = 6.35</math>; <math>p &lt; .001</math></b>
	Evaluative/ Motivational	16.81 (5.39)	15.63 (4.38)	$t(108) = 1.07$ ; $p = .287$

**Table 7** - Group data on clinical, demographic and psychological variables.

#### **4.4.2 Group differences on psychological measures**

The correlations between SCCS and the VISQ subscales were, respectively: dialogical inner speech,  $r = -.237$ ,  $p = .013$ ; condensed inner speech,  $r = -.329$ ,  $p < .001$ ; other people in inner speech,  $r = -.504$ ,  $p < .001$ ; and, evaluative/ motivational inner speech,  $r = -.293$ ,  $p = .003$ . Hence, lack of self-concept clarity was associated with low scores on all of the inner speech dimensions.

Means and standard deviations on the SCCS and the VISQ subscales for patients and comparisons, together with significance tests, are presented in Table 7. As expected, the patients scored significantly lower than the comparisons on the SCCS. Group differences were also observed for condensed inner speech and other people in inner speech but not on dialogic or evaluative/ motivational inner speech.

#### **4.4.3 Clarity of self-concept and individual psychotic experiences**

Table 8 shows the bivariate correlations between self-concept clarity and the different psychotic symptoms for our clinical sample. Poor clarity of self-concept negatively correlated with all symptoms with the exception of the negative PANSS subscale. However, our prediction was that self-concept clarity would significantly predict our TD scores even after controlling for comorbid symptoms. In order to test this prediction, we ran two two-stage linear regressions on the data from our clinical participants.

In the first regression model, we used the TD score from the non-salient interview as the dependent variable and, in order to control for co-occurring symptoms, we entered PANSS scores for hallucinatory behaviour (P3), delusions (P1) and suspiciousness/persecution (P6) in the first stage. This initial model was



significant,  $F [3, 76]= 5.19, p= .003, R^2_{\text{adjusted}}= .137$ . Adding clarity of self-concept led to a significant improvement in the model,  $F_{\text{change}} [1, 75]= 5.51, p= .022$ , leading to a significant final model,  $F [4, 75]= 5.5, p= .001, R^2_{\text{adjusted}} = .186$ , in which clarity of self-concept was a significant predictor of TD in the non-salient interview ( $b= -.261, p= .022$ ). However, delusions also remained a significant predictor ( $b= .322, p= .01$ ).

We repeated the same procedure with TD scores from the salient interview as the dependent variable. Again the first model was significant,  $F [3, 76]= 4.25, p= .008, R^2_{\text{adjusted}}= .110$ . Adding clarity of self-concept led to a significant improvement in the model,  $F_{\text{change}} [1, 75]= 6.07, p= .016$ , leading to a significant final model,  $F [4, 75]= 4.91, p= .001, R^2_{\text{adjusted}} = .165$ , in which clarity of self-concept was a significant predictor ( $b= -.277, p= .016$ ); this time none of the co-morbid symptoms remained significant.

#### **4.4.4 Inner speech and individual psychotic experiences**

In order to test the relations between the different psychotic symptoms and the four dimensions of inner speech we conducted exploratory bivariate correlations for our clinical sample (see Table 8). The only significant correlations were between other people in inner speech and hallucinations, delusions and the positive PANSS factor, and between hallucinations and evaluative and motivational speech. Condensed inner speech was correlated with TD only in the non-salient interview and dialogic inner speech was only marginally correlated with negative symptoms ( $p= .047$ ).

	<b>Dialogic inner speech</b>	<b>Condensed inner speech</b>	<b>Other people in inner speech</b>	<b>Evaluative and motivational inner speech</b>	<b>Clarity of self-concept</b>
<b>Thought disorder (non-salient)</b>	-.165	.222*	.067	-.147	-.353***
<b>Thought disorder (salient)</b>	-.163	.203	-.024	-.165	-.349**
<b>Hallucinatory behaviour (P3)</b>	.141	.102	.534***	.264*	-.307**
<b>Delusions (P1)</b>	.117	.171	.343**	.124	-.269*
<b>Suspiciousness and persecution (P6)</b>	.016	.075	.202	.054	-.294**
<b>Conceptual disorganisation (P3)</b>	-.171	.194	.038	-.184	-.375***
<b>Positive factor</b>	.114	.191	.313**	.114	-.435***
<b>Negative factor</b>	-.222*	.004	-.016	-.079	-.142

Note: \*  $p < .05$  \*\*  $p < .01$  \*\*\*  $p < .001$

**Table 8** - Bivariate correlations between TD, hallucinations, delusions, suspiciousness/persecution, negative symptoms, four dimensions of inner speech and clarity of self-concept (clinical group only).

#### 4.4.5 Dimensions of TD, inner speech, and clarity of self-concept

Because TD is a multidimensional construct (Andreasen & Grove, 1986; Berenbaum & Barch, 1995; Harvey, Earle-Boyer, & Wielgus, 1984; Peralta, Cuesta, & de Leon, 1992; Solovay et al., 1986), we decided to test how our psychological measures

related to the different dimensions of TD. In order to extract factors from the TLC, we conducted a factor analysis using the 18 TLC scores from the salient interview of our clinical group and the unweighted least squares (ULS) method with varimax rotation (given that the TLC scores did not meet the criteria for the maximum likelihood). Our factor analysis produced 6 factors with *eigenvalues* greater than 1, similar to previous findings (Cuesta & Peralta, 1999). These 6 factors explained 69.64% of the total variance and were interpreted as: *disorganised* (derailment, incoherence, illogicality, clanging, word approximations, circumstantiality, loss of goal, perseveration, and self-reference), *linguistic* (neologisms and stilted speech), *attentional* (pressure of speech and distractible speech), *poverty* (poverty of speech and tangentiality), *emptiness* (poverty of the content of speech and echolalia), and finally *blocking factor* (blocking). Table 9 shows the partial correlations between the different TD factors and clarity of self-concept after controlling for hallucinations, delusions, and suspiciousness/persecution.

Finally, in order to test the associations between the TD factors and the inner speech factors, we conducted exploratory partial correlations controlling for PANSS scores for hallucinatory behaviour (P3), delusions (P1) and suspiciousness/persecution (P6). Table 9 shows that the negative TD factor was substantially and negatively correlated with self-reported dialogic inner speech but also with other people in inner speech and evaluative and motivational inner speech. The only other significant correlation was between the linguistic TD factor and dialogic inner speech; however this was a positive correlation and marginally significant ( $p=.049$ ).

## 4.5 Discussion

The primary goal of the present study was to explore associations between clarity of self-concept (the extent to which beliefs about self are stable across time, consistent and clearly defined (Campbell et al., 1996)) self-reported inner speech, and TD. Interestingly, poor self-concept clarity was modestly associated with low scores on all of the inner speech dimensions. This finding is consistent with the suggestion by some theorists that the quality of inner speech contributes to self-knowledge and hence the coherence of the self-concept (Morin & Everett, 1990; Morin, 2007). Nonetheless, specific associations were found between self-concept clarity on the one hand and inner speech on the other and different psychotic symptoms.

As expected, our patients showed diminished clarity of self-concept. Although poor clarity of self-concept was associated with all psychotic experiences with the exception of the negative symptoms, our regression analyses revealed that it was a significant predictor of TD scores in both the salient and non-salient interviews, even after controlling for the other symptoms. Moreover, when specific TD factors were considered and co-occurring symptoms controlled for, lack of self-clarity was specifically associated with the disorganised TD factor. The findings of our study are consistent with qualitative accounts of the role of the self in TD (Lysaker & Lysaker, 2006) and complement what is already known about TD from a socio-cognitive perspective. For example, several studies have supported the association between poor ToM and TD (Corcoran, Mercer, & Frith, 1995; Frith, 1992; Hardy-Baylé et al., 2003; Sprong, Schothorst, Vos, Hox, & van Engeland, 2007).

	<b>Dialogic inner speech</b>	<b>Condensed inner speech</b>	<b>Other people in inner speech</b>	<b>Evaluative and motivational inner speech</b>	<b>Self-concept</b>
<b>Disorganised factor</b>	-.164	.208	-.075	-.185	<b>-.265*</b>
<b>Linguistic factor</b>	<b>.225*</b>	.039	.090	.184	.121
<b>Attentional factor</b>	.153	.139	.001	-.058	-.146
<b>Negative factor</b>	<b>-.457***</b>	.031	<b>-.282*</b>	<b>-.323**</b>	-.068
<b>Emptiness factor</b>	-.100	.004	-.056	.082	-.071
<b>Blocking factor</b>	-.148	-.023	.026	.061	.008
<b>Self-concept</b>	<b>-.218*</b>	-.180	-.185	<b>-.229*</b>	--

Note: \*  $p < .05$  \*\*  $p < .01$  \*\*\*  $p < .001$

**Table 9** - Partial correlations between TD factors, inner speech factors and clarity of self-concept after controlling for hallucinations, delusions, and suspiciousness/persecution (clinical group only).

It is conceivable that such difficulties could be partly explained by lack of clarity of self-concept in the patient, although this will require investigation in future studies.

The findings also provide a potential psychological mediator for research on the social origins of TD. For example, some studies have reported significant associations between childhood adversity (Shah et al., 2014; Toth, Pickreign Stronach, Rogosch,

Caplan, & Cicchetti, 2011) or institutionalization (Walker, Cudeck, Mednick, & Schulsinger, 1981) and TD. Adoption studies have reported an interaction between genetic high-risk status and family miscommunication in the long-term prediction of TD in adoptees (Wahlberg et al., 1997, 2004). It is conceivable that clarity of self-concept could play a mediating role between these risk factors and TD.

Contrary to our expectations, the only significant between-group differences on the VISQ subscales were for other people in inner speech and condensed inner speech, with the patients scoring higher on both subscales. We did not find significant differences between the groups on self-reported dialogic inner speech or evaluative and motivational inner speech. TD, from the non-salient interview only, was associated with condensed inner speech but this association was weak and barely significant.

A more complex picture of the relationship between inner speech and TD emerged when we considered the six TD factors. Controlling for other symptoms, we found that dialogic inner speech, other people in inner speech, and evaluative/motivational inner speech were all negatively correlated with our negative TD factor. The main TD item contributing to the negative factor was poverty of speech. Hence, an implication of this finding is that the absence of social speech is correlated with the absence of inner speech, an association which makes sense within the context of Vygotsky's developmental model (Vygotsky, 1934), which proposes that the ontogeny of inner speech lies in social speech. The finding of an association between poverty of speech and diminished self-reported inner speech also informs the long standing debate of whether TD is a speech or a cognitive problem (Chaika, 1982; Lanin-Kettering & Harrow, 1985).

Overall, therefore, our findings suggest that the negative and positive dimensions of TD may be associated with different psychological processes. More specifically, negative TD/poverty of speech seems to be associated with deficits in inner speech, whereas positive TD, comprising the disorganised aspects of TD, is associated with poor clarity of self-concept.

A secondary aim of the present study was to examine the relationship between inner speech and hallucinations. Other people in inner speech was significantly correlated with hallucinations and delusions in our clinical group, as was evaluative/motivational inner speech. These findings are perhaps unsurprising given that auditory hallucinations take the form of the voices of others (often others who can be identified by the hearer (Nayani & David, 1996)) and also given theoretical accounts which suggest that AVHs consist of inner speech that is misattributed to external sources (Bentall, 1990; Fernyhough, 2004; Frith, 1992). In this context, it is important to note that our participants were asked to report specifically on their inner speech rather than their voice-hearing experiences. Given previous findings indicating that schizophrenia patients can reliably distinguish their inner speech from their voices (Hoffman, Varanko, Gilmore, & Mishara, 2008; Langdon et al., 2009), the present findings can be taken to support the inner-speech model of AVHs.

The finding that the patients as a whole endorsed items relating to condensed inner speech more highly than controls suggests that schizophrenia patients' inner speech is predominantly condensed. Fernyhough (2004) has proposed that it is specifically expanded inner speech that is experienced as AVHs; the increase in condensed inner speech (which is the opposite of expanded inner speech) found in the patients in this study (although not specifically in association with hallucinations) might therefore be interpreted as consistent with this hypothesis. Possibly when inner

speech is predominantly condensed, other kinds of inner speech, (inner speech that is emotionally charged or which involves the voices of others) are especially likely to be experienced as anomalous and hence misattributed to an external source, particularly if patients also have other vulnerabilities to making these kinds of misattributions, for example impaired source monitoring (Brookwell, Bentall, & Varese, 2013).

#### **4.5.1 Limitations**

As in most studies of TD, we only recruited patients with schizophrenia spectrum diagnoses, but there is evidence that TD is a transdiagnostic construct, especially affecting patients with a bipolar diagnosis (Andreasen, 1979b; Tai et al., 2004).

Another limitation is that we used a questionnaire to quantify inner speech. The VISQ has already been used to examine sub-syndromal psychotic experiences in healthy samples (Alderson-Day et al., 2014; McCarthy-Jones & Fernyhough, 2011).

However, the methodology relies heavily on the patient's metacognitive ability to reflect about thoughts and this ability may be compromised in some patients (van der Meer, Costafreda, Aleman, & David, 2010).

In future research, one way of circumventing the limitations of the VISQ may be to complement the methodology with Descriptive Experience Sampling (Hurlburt & Akhter, 2006). This method allows for inner experience to be captured in the moment. It would also be interesting to include more comprehensive and phenomenological way of exploring self-disturbances such as the *examination of anomalous self-experience* (Parnas et al., 2005).



#### **4.5.2 Implications for clinical practice**

The most obvious implication relates to the therapeutic strategies adopted when working with thought-disordered patients. The findings seem to suggest that therapeutic work with patients who present with predominantly poverty of speech should focus on improving dialogical inner speech by perhaps promoting and incentivizing socialization and opportunities for the patient to converse. Therapeutic work with patients who present with predominantly positive TD and disorganisation should perhaps focus more on improving self-concept through consistent and coherent feedback about patients' self-knowledge and self-beliefs (Slotter & Gardner, 2014). This work should be carried out carefully given that interpersonal sensitivity seems to have an important moderating effect of TD (Grant & Beck, 2009). Lysaker and Lysaker (2002) suggest three main requirements for the rehabilitation of patient's dialogical processes, namely: a non-hierarchical relationship that promotes the patient's dialogue and self-disclosure; a commitment to helping the patient remember and explain personal views and concerns; and finally the use of strategies to promote and assist patients as they converse within themselves and with significant others about their feelings and their own representation of events.

## 4.8 References

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## **Chapter 5.   ‘*No man is an island*’: Testing the specific role of social isolation in thought disorder**

This paper has been submitted and is currently in press as de Sousa, P., Sellwood, W., Spray, A., & Bentall, R. P. (in press). ‘*No man is an island*’. Testing the specific role of social isolation in thought disorder. *Psychiatric Research*.

## 5.1 Abstract

**Background.** Recent work has focused on the role of the environment in psychosis with emerging evidence that specific psychotic experiences are associated with specific types of adversity. One risk factor that has been often associated with psychosis is social isolation, with studies identifying isolation as an important feature of prodromal psychosis and others reporting that social networks of psychotic patients are smaller and less dense than those of healthy individuals. In the present study, we tested a prediction that social isolation would be specifically associated with formal thought disorder.

**Methods.** 80 patients diagnosed with psychosis-spectrum disorder and 30 healthy participants were assessed for formal thought disorder with speech samples acquired during an interview that promoted personal disclosure and an interview targeting everyday topics.

**Results.** Social isolation was significantly associated with formal thought disorder in the neutral interview and in the salient interview, even when controlling for comorbid hallucinations, delusions and suspiciousness. Hallucinations, delusions and suspiciousness were not associated with social isolation when formal thought disorder was controlled for.

**Discussion.** *Formal* thought disorder is robustly and specifically associated with social isolation. Social cognitive mechanisms and processes are discussed which may explain this relationship as well as implications for clinical practice and future research.

## **5.2 Introduction**

Over the last decade, there has been a renewed interest in the role of social adversity in schizophrenia (Read et al., 2014; van Os et al., 2010). Factors such as familial miscommunication (de Sousa et al., 2013), migration (Cantor-Graae and Selten, 2005), exposure to urban environments (Vassos et al., 2012), childhood sexual abuse, bullying and other childhood (Varese et al., 2012b) and adulthood adverse events (Beards et al., 2013) are associated with an increase in the risk of psychosis. In addition, there is emerging evidence that specific adversities are related to specific psychotic symptoms. Examples include associations between childhood sexual abuse and hallucinations and between disrupted early attachment relationships and paranoia (Bentall et al., 2012; Shevlin et al., in press). Psychological mechanisms that might explain these relationships have also been suggested (Bentall et al., 2014; Sitko et al., 2014; Varese et al., 2012).

### **5.2.1 The relevance of formal thought disorder (FTD)**

FTD refers to a set of communicational, cognitive and language disturbances that render the speech of some individuals difficult to follow and apparently unintelligible (Andreasen, 1982). Examples of FTD can vary from instances of incoherence (e.g. “Yes, they add up and kind of like a solution. It’s say, it’s a equine or equinox, like fungi. Something in the brain tells you it’s a high number. Bacteriology, a numerate number, it’s a particle, therefore it contains solution is to answer the right question” Laws et al., 1999, p. 105) to illogicality (e.g. “Parents are the people that raise you. Anything that raises you can be a parent. Parents can be anything, material, vegetable, or mineral, that has taught you something” Andreasen, 1986, p. 478).

These disturbances have been relatively neglected in social psychiatry research but are important for several reasons. First, FTD is highly prevalent in psychotic patients, with some estimates reaching 91% (Roche et al., 2014). Second, it is associated with poorer occupational functioning (Racenstein et al., 1999), poorer social functioning (Bowie and Harvey, 2008; Bowie et al., 2011), and poorer quality of life (Tan et al., 2014). Third, FTD has been found to be highly predictive of future psychotic relapse (Wilcox, 1990) a picture that is further complicated by the relative lack of evidence-based therapeutic strategies to address it (Beck et al., 2009; Stolar and Grant, 2011) and its persistent course (Bowie et al., 2005; Docherty et al., 2003; Marengo and Harrow, 1987, 1997). Last but not least, FTD seems to be an early predictor of later conversion into psychosis in high-risk populations (Bearden, et al., 2011; Cannon et al., 2008; Ott et al., 2002) providing clinicians and services alike with a potential window of opportunity for early detection and preventative work.

### **5.2.2 Psychological mechanisms in FTD**

Over the years several psychological mechanisms have been evoked to explain FTD including difficulties at the level of ‘theory-of mind’ (ToM; Frith, 1992; Hardy-Baylé et al., 2003; Sprong et al., 2007), poor internal source monitoring (Harvey, 1985; Nienow and Docherty, 2004), deficits at the level of executive function (Kerns and Berenbaum, 2002; McGrath, 1991) and semantic hyperpriming (Pomarol-Clotet et al., 2008; Spitzer, 1997). A widely replicated finding, reported in both schizophrenia patients and bipolar patients, is that FTD and communication disturbances are more evident when patients discuss affective-laden topics (Docherty et al., 1994; Docherty, 1996, 2005; Haddock et al., 1995; Shimkunas, 1972; Tai et al., 2004).

Much less is known about social predictors of FTD. Although FTD has often been assumed to be an endophenotype of schizophrenia (Levy et al., 2010; Meehl, 1962) several studies have identified important psychosocial factors associated with its development such as dysfunctional family communication (Roisko et al., 2014; Wahlberg et al., 1997, 2000), childhood adversity (Shah et al., 2014; Toth et al., 2011) and institutionalization (Walker et al., 1981).

### **5.2.3 Social isolation and psychosis**

Since Faris and Dunham's (1939; Faris, 1934) classic ecological study in Chicago, there has been an accumulation of studies showing that social isolation is an important factor in psychosis (Boydell et al., 2004; Van Os et al., 2000). The relevance of social isolation in schizophrenia has also been well acknowledged in the psychoanalytical literature (Sullivan, 1953). For example, Freud argued for the centrality of the patient's withdrawal from the surrounding world as a crucial process in psychosis (i.e. process of libidinal decathexis, Freud, 1914) and other authors have argued that this process of desocialisation is crucial to understand psychotic experiences given its detrimental impact on symbolic thought (Arieti, 1955).

Consistent with this, early empirical studies have reported for example higher levels of social isolation in communities with high incidence rates of schizophrenia (Jaco, 1954) and higher rates of social isolation in patients diagnosed with schizophrenia (Hirschberg, 1985). These findings have been supported by other studies that have reported that psychotic patients have smaller social networks (Erickson et al., 1989), fewer individuals in their social networks (Macdonald et al., 2000), fewer confidants (Morgan et al., 2008) and are three times more likely to have low frequency of contact with others in their social network (Reininghaus et al., 2008)

with some studies suggesting that this may be significantly more pronounced in urban environments (Schomerus et al., 2007).

Population studies with a non-clinical samples have also reported associations between lack of perceived social support and psychotic experiences (Alptekin et al., 2009) and a dose-response relationship between having smaller primary network at baseline and self-reported psychotic experiences at 18-month follow-up (Wiles et al., 2006). Other studies and reviews have reported that isolation is also a factor that challenges patient's recovery (Soundy et al., 2015), is associated with increased number of admission (Simone et al., 2013) and with poorer outcomes (Harvey et al., 2007).

It has been suggested that social isolation may be the result of a "social network crisis" following first admission to a mental health ward (Lipton et al., 1981). However, the population studies mentioned above have been carried out with samples of non-clinical participants that have never been admitted. Moreover, both retrospective and prospective birth cohort studies have found that social isolation in childhood is associated with a later diagnosis of schizophrenia (Cannon et al., 1997; Jones et al., 1994; Welham et al., 2009). In a cohort study of 50,054 Swedish conscripts, individuals who later developed psychotic experiences at a 15-year follow up were significantly more likely to have fewer than two friends and to prefer smaller groups (Malmberg et al., 1998) suggesting that social isolation may predate the onset of symptoms and the diagnosis. Consistent with this, a recent systematic review revealed that individuals diagnosed with first episode of psychosis have significantly smaller social networks than healthy individuals suggesting again that social isolation and smaller social networks predate onset of psychotic disorder (Gayer-Anderson and Morgan, 2013). Finally, studies of individuals with prodromal symptoms report as

well that social withdrawal is very common feature in individuals before the onset of psychosis (Tan and Ang, 2001; Mäki et al., 2014).

#### **5.2.4 Social isolation and specific symptoms**

How might social isolation contribute to the onset, development or maintenance of individual psychotic symptoms? Hoffman (2007, 2008) has suggested that social isolation and withdrawal during critical developmental periods may lead to deafferentation of brain regions that support social cognition and therefore predispose individuals to psychotic experiences (e.g. leading to the induction of anomalous experiences). Studies using animal models have reported findings consistent with this hypothesis (Fabricius et al., 2011; Fone and Porkess, 2008; Silva-Gómez et al., 2003).

In a more psychological account, Freeman and colleagues (Freeman et al., 2002; Freeman and Garety, 2006; Freeman, 2007) have suggested that social isolation may contribute to maintenance of persecutory beliefs by not allowing opportunities for these beliefs to be reviewed and disconfirmed by people in the social network of the individual. Drawing on data from large population study, Freeman and colleagues (2011) reported an association between self-reported paranoia and a range of demographic (e.g. being single) and psychological indicators of social isolation (e.g. less perceived social support). However, in a different study the association between number of social supports and paranoia was not significant when authors adjusted for confounders (Freeman et al., 2008).

The possible association between FTD and social isolation has not yet been explored empirically but there are some interesting clues to why isolation might be a particularly relevant factor in this cluster of symptoms. Some authors have reported that, when thought-disordered patients are asked to clarify some of their utterances,



for example by providing more contextual information, these utterances become intelligible and comprehensible (Harrow et al., 1983). Hence, patients seem able to construct coherent utterances when cued to do so in an appropriate social context. The same group of researchers proposed that patients' apparent unintelligible utterances may be a consequence of the *intermingling* of decontextualized personal concerns and worries coupled with an inability to take the perspective of the listener and to speak to the listener's needs (Harrow et al., 1983, 2000; Lanin-Kettering and Harrow, 1985) which is a prerequisite for the establishment of conversational alignment (Pickering and Garrod, 2006). Such an account is consistent with those social-cognitive models of FTD (Frith, 1992; Hardy-Baylé et al., 2003; Sprong et al., 2007) that propose that the thought disordered individual is unable to adjust speech to the needs of an audience due to a failure to represent the state of knowledge of the listener.

One possible explanation for the FTD is that an inability to take the perspective of the listener may be due to a lack of social contact and consequentially missed opportunities for social feedback during conversation (Hammer et al., 1978); after all, conversation is a social skill. To the best of our knowledge, this hypothesis has never previously been tested but it is consistent with evidence from studies that have reported that communicability in FTD participants can be improved through video-taped feedback (Satel and Sledge, 1989).

### **5.2.5 Aims of the present study**

In the present study, we therefore test whether participants diagnosed with psychotic-spectrum disorders report significantly more social isolation than non-clinical participants and more importantly, if levels of social isolation specifically predict scores of FTD. Given previous arguments that hallucinations (Hoffman, 2007, 2008)

and paranoia (Freeman et al. 2002; Freeman, 2007) are associated with social isolation, we also studied the associations between social isolation and these symptoms, in each case controlling for the presence of comorbid FTD.

## **5.3 Methods**

### **5.3.1 Participants and procedures**

80 clinical participants (see Table 10) were recruited from local mental health sites across the North West of England. Participants were identified and referred by local care coordinators such as mental health nurses, social workers and consultant psychiatrists. The recruitment targeted 18-65 year olds with a psychotic-spectrum disorder as primary diagnosis defined as schizophrenia (F20), schizoaffective (F25) or unspecified non-organic psychosis (F29) according to ICD-10 (World Health Organization, 2004). We excluded participants who lacked capacity for informed consent (as determined by care coordinator) whose first language was not English as well participants with severe learning difficulties; recent alcohol or drug abuse; history of neurological disorders or any other non-psychotic disorders that could affect brain function (only two potential participants were excluded – one because his first language was not English and the other because patient was deemed by own care team to be too unwell to take part in the study). Demographic and clinical information was taken from participant during the first visit or from members of the care team (almost always the mental health practitioner responsible for the patient's care e.g. mental health nurse or social worker) with participant's prior consent. Antipsychotic

		Patients	Comparisons	
Number		80	30	
Single (%)		67 (83.7%)	15 (50%)	$\chi^2 (1) = 13.10, p < 0.001$
Unemployed (%)		77 (96.3%)	11 (36.7%)	$\chi^2 (1) = 48.41, p < 0.001$
Social isolation (LSNS-18)		60.2 (16.3)	40.8 (13.7)	$t (108) = 5.80, p < 0.001$
Sex (%)	Male	58 (72.5%)	21 (70%)	$\chi^2 (1) = 0.07, p = 0.795$
	Female	22 (27.5%)	9 (30%)	
Ethnicity (%)	White British	74 (92.5%)	28 (93.3%)	$\chi^2 (1) = 0.02, p = 0.881$
	White Irish	2 (2.5%)	1 (3.3%)	
	Black British	3 (3.8%)	0 (0%)	
	Other	1 (1.3%)	1 (3.3%)	
Age (years)		39.3 (11.6)	38.4 (13.3)	$t (108) = 0.33, p = 0.746$
Years of education		11.2 (1.9)	12.7 (2.3)	<b><math>t (108) = -3.35, p = 0.001</math></b>
IQ		98.4 (10.6)	109.5 (8.3)	<b><math>t (108) = -5.18, p &lt; 0.001</math></b>
Diagnoses (%)	Schizophrenia (F20)	48 (60%)	N/a	
	Schizoaffective (F25)	18 (22.5%)	N/a	
	Other Psychoses (F29)	14 (17.5%)	N/a	

Duration of illness (years)	15.2 (10.9)	N/a
History of admission (yes)	73 (91.3%)	N/a
History of Mental Health act (yes)	62 (77.5%)	N/a
First-generation antipsychotics (%)	26 (23.6%)	0 (0%)
Second-generation antipsychotics (%)	58 (72.5%)	0 (0%)
‘Mood stabilizers’ (%)	14 (17.5%)	0 (0%)
Anti-depressants (%)	31 (38.7%)	0 (0%)
Equivalent CPZ dose (mg)	469.7 (389.1)	N/a
PANSS	Positive	17.1 (5.2)
	Negative	14 (4.7)
	General	38.6 (9.2)
	Total	69.8 (16.1)

**Table 10** - Clinical and demographic variables.

medications were converted to chlorpromazine equivalents as per agreed conventions (Woods, 2003).

For comparison purposes, 30 participants with no history of psychosis were recruited through local advertisements in the community (also shown in Table 10). All of these participants were screened for psychotic symptoms with the Psychosis Screening Questionnaire (PSQ; Bebbington and Nayani, 1995). An attempt was made to select participants who were approximately comparable with our participants in the clinical group on variables such as sex, age and ethnicity. None of the non-clinical participants were excluded (see appendix 1 for correspondence regarding ethical approval and appendices 2, 3 and 4 for consent form and information sheets).

The current study is a case-control study that is part of a larger research project on the social, cognitive and affective predictors of FTD. The research project was approved by UK National Research Ethics Service (NRES), by the R&D departments of local NHS Mental Health Trusts (Merseycare NHS Trust and Cheshire and Wirral Partnership) and was sponsored by the University of Liverpool.

All participants were provided with information about the study and allowed a week to decide whether they wanted to take part. After consenting, all participants were seen twice on different days. The interval between the two sessions was in most cases a few days and never more than one week. Participants in the clinical group were interviewed with PANSS (Kay et al., 1987) whereas controls were screened with the PSQ (Bebbington and Nayani, 1995; see appendix 6) all the interviews were carried out by the first author and took on average 30-45 minutes. Following these assessments, participants then completed the QT and LSNS-18 (see below). All interviews and testing took place in the participants' homes with the exception of

three participants who were interviewed at the University of Liverpool as per individual request. Each participant was interviewed using the salient and non-salient interviews (Haddock et al., 1995; Tai et al., 2004) in a randomly counterbalanced order across the two sessions. Each interview followed a strict protocol and each question started with the same statement (e.g. “Can you tell me about...?”). Interviews lasted approximately 15 minutes on average, providing authors with 30-minutes of speech per participant. Each participant was paid £20 for participation.

The speech samples were recorded with a digital voice recorder (Olympus VN711 PC 2GB) and later transcribed by the first author and a professional transcriber, before being coded by PS and AS using the TLC.

The first and third authors independently coded 10% (22) of the speech samples for reliability purposes. The coding was preceded by the careful reading of the TLC and relevant papers (Andreasen, 1979a, 1979b, 1986; Andreasen and Grove, 1986) and by practice sessions. For some of the TLC items it was not possible to calculate a Kappa, as they were very infrequent (e.g. neologisms, clanging, etc.). For the remaining items all Kappa values were of substantial magnitude with tangentiality achieving the highest level of agreement ( $K=0.82$ ) and self-reference the lowest ( $K=0.62$ ).

### **5.3.2 Assessment tools**

#### ***2.3.2.1 Psychotic symptoms***

Psychotic symptoms were measured using the *Positive and Negative Syndromes Scale* (PANSS; Kay et al., 1987; see appendix 5). The PANSS is a widely used clinical interview that measures 30 symptoms, comprising a positive symptom scale, a negative symptom scale, and a general psychopathology scale. Each item is scored

from 1 to 7 with the higher score indicating increased severity. The scale has been found to have good psychometric properties (Kay et al., 1987). The means and standard deviations for the clinical group are presented in Table 10.

#### **5.3.2.2. IQ**

Pre-morbid verbal intelligence was measured using the Ammon's Quick test (QT; Ammons and Ammons, 1962; see appendix 7), a picture-vocabulary test, which is not timed and therefore ideal for the study. The participant is presented with four pictures of different situations (e.g. a policeman stopping the traffic with a whistle so that two schoolchildren can cross the road) and is asked to identify fifty progressively difficult words by simply pointing to the appropriate card where the word referent can be found (e.g. "whistle"). The final score is achieved by summing the number of words correctly identified by the participant. The QT has been extensively used in clinical studies with mental health participants and correlates with WAIS scores (Lezak, 2004). All QT scores were converted into IQ equivalent scores using standardised guidelines. The means and standard deviations for both groups are presented in Table 10.

#### **5.3.2.3 Interviews**

Speech samples were gathered from all participants using two interview protocols that had been previously developed to elicit FTD (Haddock et al., 1995; Tai et al., 2004; see appendix 8). The protocols elicited speech samples relating to emotionally-laden (salient interview) and neutral topics (non-salient interview), given the evidence that participants diagnosed with psychosis show more FTD when asked to talk about emotional topics (Docherty et al., 1994; Docherty, 2005; Shimkunas, 1972). The

salient interview involved fifteen questions (and eight reserve questions) that promoted self-disclosure by asking for negative autobiographical memories (e.g. “Can you tell me about the most awful thing that someone has done to you?”), whereas the neutral interview included fifteen questions (and six reserve questions) that did not promote self-disclosure (e.g. “Can you tell me about travelling on public transports?”). Mean duration of the interviews as well as means and standard deviations of the different word counts are presented in Table 11.

#### **5.3.2.4 FTD**

The speech samples were rated using the Scale for the Assessment of Thought, Language and Communication (TLC; Andreasen, 1986; see appendix 9), a widely used scale that provides definitions and scores for 18 different types of FTD. Some items are considered more pathological and others less pathological. The different categories of FTD are rated on a scale of severity ranging from 0 to 4 or 0 to 3 (depending on the item). The global rating is achieved by summing the scores of the different subscales (with the score of the more pathological items being multiplied by 2). The scale can be applied to any speech samples and has been shown to have good psychometric properties (Andreasen, 1979b, 1986).

#### **5.3.2.5 Social Isolation**

Social isolation was measured with the *Lubben Social Network Scale* (LSNS-18; Lubben, 1988; see appendix 12), a self-report questionnaire that measures the size, closeness and frequency of contacts within social network using 18 items (e.g. “How many relatives do you see or hear from at least once a month?”). The scores for each question range from 0 to 5 with the higher score representing more social isolation



(we reversed the original scoring for purposes of simplicity). The questionnaire is divided across three social network domains (family, neighbours and friends) and the instrument has been found to have good internal consistency (Lubben and Gironde, 2004). The highest possible total score is 90 and the lowest score is obviously 0. Table 10 shows the mean and standard deviation of the scores for both groups.

### **5.3.3 Statistical analysis**

Statistical analyses were carried out on IBM SPSS Statistics. T-tests and Chi-squares were used to characterize and compare the groups on demographic variables and social isolation. Interrater reliability for TLC scores (FTD) was calculated using Cohen's kappa coefficients for the different TLC items. We tested the differences in the frequency of the different TLC items between groups for both interviews using a MANOVA with Bonferroni correction for multiple comparisons as suggested in the literature (Sainani, 2009). A 2x2 mixed ANOVA was used to compare FTD variables between groups and across conditions. We used partial correlations to explore relationships between variables controlling for age, sex, education and verbal IQ (Bowie et al., 2005; Roche et al., 2014) with and without Bonferroni correction. Finally, to test whether FTD was significantly and specifically predicted by social isolation we conducted two independent two-stepped linear regressions with the FTD scores of both interviews (neutral and salient interview) as the outcome variable. In both regression models we entered the PANSS scores for hallucinations, delusions and suspiciousness in the first step and added the social isolation score in the second step. Finally, to complement the statistical analyses, we conducted three further two-stepped linear regressions using PANSS scores for delusions, hallucinations and suspiciousness as dependent variables. In these, FTD scores and remaining symptoms

	Neutral			Salient		
	Patients	Comparisons	<i>F</i> (2, 109)	Patients	Comparisons	<i>F</i> (2, 109)
Poverty of speech	30 (37.5%)	0 (0%)	<b>13.25</b> <sup>***</sup>	40 (50%)	1 (3.3%)	<b>18.90</b> <sup>***</sup>
Poverty of content of speech	1 (1.3%)	0 (0%)	0.37	9 (11.3%)	0 (0%)	3.29
Pressure of speech	7 (8.8%)	0 (0%)	2.49	8 (10%)	0 (0%)	2.87
Distractible speech	13 (16.3%)	2 (6.6%)	1.79	15 (18.8%)	1 (3.3%)	4.26 <sup>*</sup>
Tangentiality	50 (62.5%)	4 (13.3%)	<b>26.52</b> <sup>***</sup>	59 (73.8%)	10 (33.3%)	<b>23.32</b> <sup>***</sup>
Derailment	34 (42.5%)	2 (6.6%)	<b>12.89</b> <sup>***</sup>	36 (45%)	6 (20%)	9.00 <sup>**</sup>
Incoherence	7 (8.8%)	1 (3.3%)	1.18	18 (22.5%)	3 (10%)	3.17
Illogicality	25 (31.3%)	4 (13.3%)	5.05 <sup>*</sup>	36 (45%)	9 (30%)	4.70 <sup>*</sup>
Clanging	3 (3.8%)	0 (0%)	1.15	5 (6.3%)	0 (0%)	1.96
Neologisms	2 (2.5%)	0 (0%)	0.76	3 (3.8%)	0 (0%)	1.15
Word approximations	27 (33.8%)	6 (20%)	2.77	26 (32.5%)	8 (26.6%)	1.73
Circumstantiality	15 (18.8%)	10 (33.3%)	0.49	27 (33.8%)	15 (50%)	0.01
Loss of goal	17 (21.3%)	1 (3.3%)	5.04 <sup>*</sup>	22 (27.5%)	6 (20%)	1.12
Perseveration	9 (11.3%)	0 (0%)	3.30	29 (36.3%)	6 (20%)	4.72 <sup>*</sup>
Echolalia	0 (0%)	0 (0%)	-	2 (2.5%)	0 (0%)	0.76
Blocking	5 (6.3%)	0 (0%)	1.76	10 (12.5%)	2 (6.6%)	0.89
Stilted speech	4 (5%)	0 (0%)	0.33	5 (6.3%)	0 (0%)	1.46
Self-reference	16 (20%)	1 (3.3%)	4.76 <sup>*</sup>	12 (15%)	0 (0%)	4.53 <sup>*</sup>

Words spoken	1388 (556.3)	2046.4 (315.1)	37.37***	1424.5 (622.6)	2042.9 (359.1)	24.8***
Duration of interview	00:15:04	00:15:03	0.0	00:15:15	00:15:02	0.28
Formal thought disorder	8.16 (6.143)	1.77 (2.019)	31.09***	12.35 (9.312)	3.93 (2.791)	22.12***

Note: \*  $p < 0.05$  \*\*  $p < 0.01$  \*\*\*  $p < 0.001$ ;

Values highlighted in **bold** represent significance after Bonferroni correction  $p < .0028$  (alpha = 1–0.95).

**Table 11** - Frequencies and percentages of the TLC items across groups and interviews with group comparisons (with and without Bonferroni corrections).

were entered in the first step as control variables and social isolation was added in the second step.

## 5.4 Results

### 5.4.1 Demographics and clinical variables

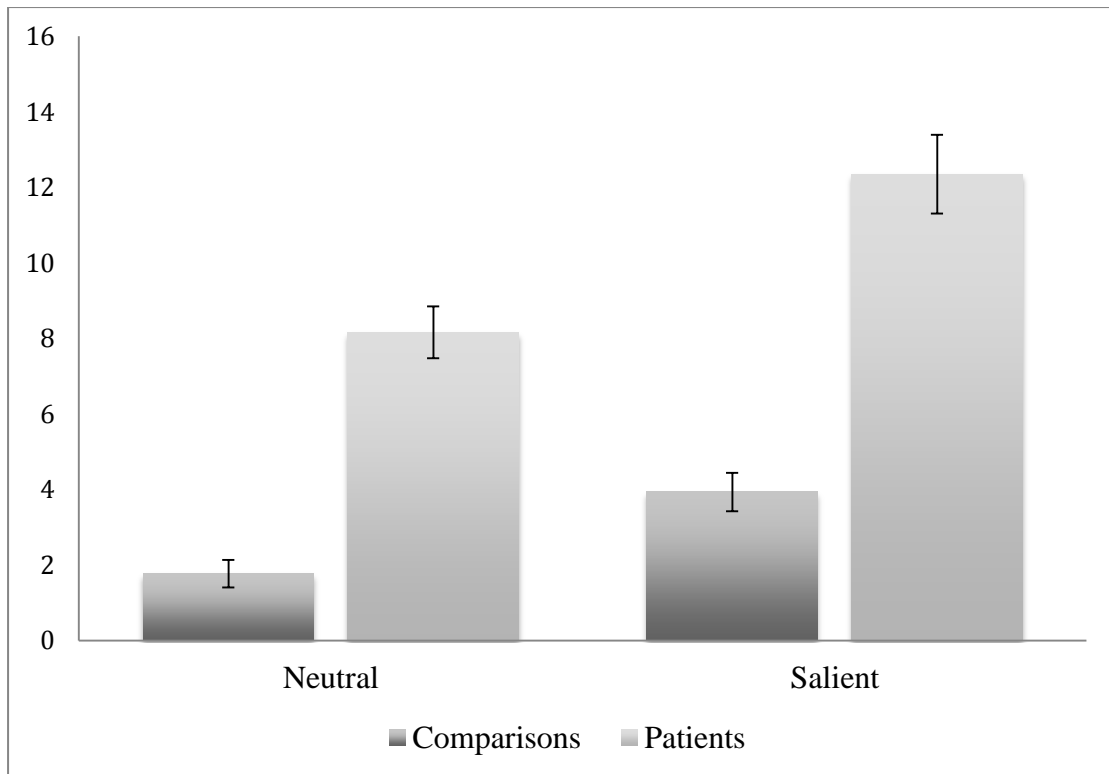
Table 10 shows descriptive statistics for some of the demographic and clinical measures. The two groups did not differ for sex ( $\chi^2 = 0.07$ ;  $p = 0.795$ ), age ( $t = 0.33$ ;  $p = 0.746$ ) or ethnicity ( $\chi^2 = 0.01$ ;  $p = 0.936$ ). However, our comparison group had significantly more years of education ( $t = -3.35$ ;  $p < 0.001$ ) and significantly higher scores on the Quick test ( $t = -5.18$ ;  $p < 0.01$ ). The means and standard deviations of the PANSS factors approximate to the values reported in other studies (Kay et al., 1987). More participants in our clinical group were single and unemployed than in the comparison group ( $\chi^2 = 11.38$ ;  $p < 0.001$  and  $\chi^2 = 44.76$ ;  $p < 0.001$ , respectively).

### 5.4.2 FTD

Table 11 displays the distribution of FTD scores across the two groups and interviews with between group comparisons for the individual TLC items. One-way MANOVAs based on all of the individual TLC items showed significant differences between groups in both salient ( $F[18,91] = 2.67$ ,  $p < 0.001$ ,  $\eta p^2 = 0.35$ ) and non-salient interviews ( $F[17,92] = 2.84$ ,  $p < 0.001$ ,  $\eta p^2 = 0.34$ ). After Bonferroni correction, patients displayed significantly more FTD only on poverty of speech (marked reduction in speech), tangentiality (these two items in both interviews) and derailment in the neutral interview. However, it should be noted that the Bonferroni method is a highly conservative test (*see* Sainani, 2009). The frequency of the different items in both participants and comparisons comes very close to the distributions of scores

originally reported by Andreasen and Grove (1986) with the exception that we found more instances of circumstantiality. This may well be due to the nature of our protocol, which invited participants to speak about emotionally challenging topics. TLC categories such as circumstantiality (a pattern of speech that is delayed getting to the point and that is marked by excessive and irrelevant details), illogicality (a pattern of speech marked by inferences that are illogical) or tangentiality (speaker replies to a question in a way that is only vaguely related to the topic) could just reflect that the participant found it hard to answer the emotionally-salient question or even avoided it by “going off on a tangent”. Moreover, it is interesting to note that we found evidence of attenuated FTD amongst healthy volunteers, especially in the salient condition, replicating Andreasen’s original findings (Andreasen, 1979a; Andreasen and Grove, 1986).

In order to compare FTD between groups and across conditions (salient and non-salient interview), we conducted a 2x2 mixed ANOVA using TLC total scores. There was a non-significant interaction between group and condition,  $F[1, 108]=3.88, p=0.052, \eta p^2=.04$ . There was substantial main effect for condition,  $F[1, 108]=38.33, p<0.001, \eta p^2=0.26$  with both groups showing an increase in FTD in the salient condition (see Table 12). The main effect comparing the two groups was also significant,  $F[1, 108]=28.93, p<0.001, \eta p^2=0.21$ .



**Table 12** - Means and standard errors for formal thought disorder (axis Y) scores in patients and comparisons across both neutral and salient interviews.

### 5.4.3 Social Isolation

The means and standard deviations for the social isolation scores are presented in Table 10. As expected, the clinical group was significantly more isolated than our comparison group ( $t= 5.80$ ;  $p< 0.001$ ).

### 5.4.4 Social Isolation and symptoms

FTD in the comparison group was not significantly correlated with social isolation in either the salient ( $r= 0.21$ ,  $p= 0.269$ ) or the non-salient condition ( $r= 0.04$ ;  $p= 0.818$ ). Table 13 shows the partial correlations between social isolation, FTD in both salient and non-salient interviews, hallucinations (P3), delusions (P1), suspiciousness (P6) and conceptual disorganization (P2) for our 80 clinical participants controlling for

sex, age, years of education and verbal IQ. It is worth noting the lack of significant associations between social isolation and delusions, hallucinations, suspiciousness and the modest association with the PANSS negative factor as opposed to the strong association with FTD in both interviews. Also, relevant is the robust and significant associations between FTD on both interviews and conceptual disorganisation item of the PANSS (P2). These p-values survived Bonferroni correction.

In order to explore the relationships between symptoms and social isolation in more detail we conducted five regression analyses using data from our clinical sample only. Control symptoms were entered in the first step and then isolation scores were entered in a second step (see Table 14). In the analysis with FTD from the salient interview as the dependent variable, the initial model with the other symptom predictors (hallucinations, delusions and suspiciousness) was significant. Adding social isolation improved the model, leading to a final significant model, in which, social isolation was a significant predictor of FTD ( $b = 0.52, p < 0.001$ ).

In the second analysis, we used FTD from the neutral interview as the dependent variable. The initial model with the other symptom predictors (hallucinations, delusions and suspiciousness) was significant. Adding social isolation improved the model, leading to a final significant model, in which, social isolation was a significant predictor of FTD ( $b = 0.46, p < 0.001$ ).

In three further analyses, we used PANSS P1 (delusions), P3 (hallucinations) and P6 (suspiciousness) as dependent variables using both FTD scores (salient and non-salient) as well as the remaining symptoms as control variables in the first step. In each case, the addition of social isolation in the second step failed to improve the model,  $F_{\text{change}} [1, 74] = 0.3, p > 0.5$  for all analyses, and social isolation failed to predict the symptom in the final model.

	Social Isolation	Hallucinations (P3)	Suspiciousness (P6)	Delusions (P1)	Conceptual disorganization (P2)
<b>Social isolation</b>	-	0.02	0.22	0.15	<b>0.52<sup>***</sup></b>
<b>Formal thought disorder (Non-salient)</b>	<b>0.43<sup>***</sup></b>	0.13	0.26 <sup>*</sup>	0.31 <sup>**</sup>	<b>0.65<sup>***</sup></b>
<b>Formal thought disorder (salient)</b>	<b>0.54<sup>***</sup></b>	0.06	0.34 <sup>**</sup>	0.24 <sup>*</sup>	<b>0.79<sup>***</sup></b>

Note: <sup>\*</sup>  $p < 0.05$  <sup>\*\*</sup>  $p < 0.01$  <sup>\*\*\*</sup>  $p < 0.001$

Values highlighted in **bold** represent significance after Bonferroni correction  $p < 0.004$  (alpha = 1–0.95)

**Table 13** - Partial correlations (Pearson's  $r$ ) between psychotic symptoms, PANSS factors, formal thought disorder and social isolation after controlling for gender, age, years of education and verbal IQ (with and without Bonferroni correction).



## 5.5 Discussion

Consistent with previous studies, the results of the present study show that our clinical participants reported significantly more social isolation than our non-clinical controls (Hirschberg, 1985; Erickson et al., 1989; Macdonald et al., 2000; Reininghaus et al., 2008). Secondly, and more importantly social isolation was found to be strongly and specifically associated with FTD even when comorbid psychotic experiences such as hallucinations, delusions and suspiciousness and potential important confounders such as sex, age, years of education and verbal IQ were accounted for (Gayer-Anderson and Morgan, 2013). That social isolation did not predict either hallucinations or delusions was unexpected given that isolation has been theorized to be implicated in their development and maintenance (Freeman, 2007; Hoffman, 2007, 2008) but again and as discussed in the introduction the association between paranoia and social isolation has been proven to be weak at least in non-clinical samples (Freeman et al., 2008). In this study, we also replicated previous findings showing that FTD worsens when patients discuss emotionally-laden topics i.e. emotional reactivity of speech (Docherty et al., 1994).

It is also interesting to note the significant association between FTD and delusions but not between FTD and hallucinations (although this association did not survive Bonferroni correction). One possible explanation for this pattern of co-occurrence is that the personal worries and concerns that are at the core of delusional beliefs may be the same decontextualized worries and concerns that intermingle and intrude in the thought disordered patient's speech as suggested by Harrow and colleagues (Harrow et al., 1983; Harrow and Quinlan, 1985; Lanin-Kettering and Harrow, 1985).

Predictors		B (S.E.)	95% CI		<i>b</i>	Model Summary
Salient interview	Hallucinations (P3)	-0.18 (0.68)	-1.54	1.18	-0.03	$F(3,76)=4.25^{**}$ $R^2_{\text{adjusted}}=0.11$
	Delusions (P1)	1.39 (0.78)	-0.15	2.93	0.23	
	Suspiciousness (P6)	1.47 (0.79)	-0.10	3.05	0.22	
	Hallucinations (P3)	-0.25 (0.58)	-1.41	0.90	-0.04	$F(4,75)=12.13^{***}$ $R^2_{\text{adjusted}}=0.36$
	Delusions (P1)	0.74 (0.67)	-0.59	2.07	0.12	
	Suspiciousness (P6)	0.99 (0.68)	-0.36	2.34	0.15	
	<b>Social isolation</b>	<b>0.29 (0.05)</b>	<b>0.19</b>	<b>0.41</b>	<b>0.52^{***}</b>	
Predictors		B (S.E.)	95% CI		<i>b</i>	Model Summary
Neutral Interview	Hallucinations (P3)	0.22 (0.44)	-0.67	1.10	0.06	$F(3,76)=4.25^{**}$ $R^2_{\text{adjusted}}=0.11$
	Delusions (P1)	1.36 (0.50)	0.36	2.36	0.34^{**}	
	Suspiciousness (P6)	0.40 (0.52)	-0.62	1.43	0.09	
	Hallucinations (P3)	0.18 (0.39)	-0.60	0.96	0.05	$F(4,75)=10.72^{***}$ $R^2_{\text{adjusted}}=0.33$
	Delusions (P1)	0.98 (0.45)	0.09	1.88	0.25^{*}	
	Suspiciousness (P6)	0.12 (0.46)	-0.79	1.03	0.03	
	<b>Social isolation</b>	<b>0.17 (0.04)</b>	<b>0.10</b>	<b>0.25</b>	<b>0.46^{***}</b>	

Note: \*  $p < 0.05$  \*\*  $p < 0.01$  \*\*\*  $p < 0.001$

**Table 14** - Two-stepped regressions with formal thought disorder (n= 80) from both interviews as the outcome and hallucinations (P3), delusions (P1), suspiciousness (P6) and social isolation as predictors.

The present findings can be contextualized in a larger endeavour to link specific adversities to psychotic symptoms (Bentall et al., 2012; Varese et al., 2012) and are especially important given our currently poor understanding of the role of social context and the environment in FTD. Obviously, this cross-sectional study cannot answer the direction of causality, and it remains possible that FTD leads to social isolation. In this context, it is interesting to note that social isolation has been found to predate the onset of psychosis in birth cohort (Jones et al., 1994; Welham et al., 2009) and prodromal studies (Malmberg et al., 1998; Tan and Ang, 2001). In addition, it could be argued that, if social isolation was a consequence of psychosis we should expect to observe stronger associations with hallucinations and delusions. Furthermore, some researchers have reported a significant association between deactivating attachment strategies and FTD in patients diagnosed with schizophrenia and bipolar disorder (Dozier and Lee, 1995). Deactivating strategies are employed when proximity seeking is perceived as dangerous and they help to maintain psychological distance, suppress attachment-related needs, avoid intimacy, emotional involvement and self-disclosure (Shaver and Mikulincer, 2007) which could potential explain why our participants showed more FTD in the interview that promoted personal disclosure.

It has been argued that one of many criteria for inferring causality in non-experimental studies is the identification of plausible mechanisms (Hill, 1965). This issue is especially relevant given the robustness and specificity of the association we report. Why would social isolation be so toxic for the communication, cognitive and linguistic skills of the psychotic individual? Given our current knowledge of the psychological processes and mechanisms involved in TD, one plausible explanation is that social isolation affects aspects of social cognition such as ToM (Corcoran et al., 1995; Docherty et al., 2013; Frith, 1992; Hardy-Baylé et al., 2003; Sprong et al., 2007) and internal source monitoring (Harvey, 1985; Nienow and Docherty, 2004) which have both being implicated in this cluster of symptoms.

Perhaps a lack of social interaction and conversational opportunities has a detrimental impact on the individual's capacity to successfully align and share topics. This impact is felt through an effect on social cognitive mechanisms (e.g. ToM, emotion perception, social perception and social knowledge) known to be impaired in psychotic participants (Savla et al., 2013) and which are important for effective communication. Interestingly, the same social-cognitive difficulties have also been reported in the relatives of participants diagnosed with schizophrenia (Lavoie et al., 2013) and are consistent with patterns of family miscommunication found in parents of psychotic participants (de Sousa et al., 2013).

### **5.5.1 Limitations**

There are several limitations to the present study. The most obvious, already noted, is that we used a cross-sectional design making it very difficult to disentangle cause and effect. Furthermore, social isolation develops and changes across time and this dynamic factor is very difficult to capture with such a design.

Another important limitation of the present study is the use of a self-report questionnaire to measure the participants' social networks. These measures are obviously open to distortions and recall biases that are extremely difficult to control. Also, we opted to recruit only participants diagnosed with psychotic-spectrum disorders but there is also evidence that FTD is a transdiagnostic cluster of experiences that is highly prevalent in patients diagnosed with bipolar affective disorder (Andreasen and Grove, 1986; Tai et al., 2004) amongst other diagnoses (McKenna and Oh, 2005).

In a future study, it would be interesting to investigate the relationship between social isolation and FTD across time using a longitudinal design and employing more robust measures of social isolation (e.g. a standardised interview where non-specific prompting can be used and collateral information can be gathered from significant others). It may also be

useful to use a transdiagnostic framework by including participants with other diagnoses (e.g. bipolar affective disorder).

Finally, in our study we only controlled for hallucinations, delusions and suspiciousness in future studies it would be relevant to include specific measures for other symptoms (e.g. anxiety or negative symptoms) and measures targeting specific psychological factors associated with psychotic experiences (e.g. degree of conviction of delusional belief).

### **5.5.2 Clinical implications: From the lab to therapy**

Further exploration of the psychological mechanisms mediating between life circumstances and psychosis and especially FTD may have important implications for clinical care. At present, there are no evidence-based strategies to address FTD but the identification of the responsible mechanisms could lead to the development of targeted interventions that can be tested in clinical trials (e.g. specific training in conversational alignment with sensitive feedback when speech is difficult to follow). As reported elsewhere in this paper some authors have provided some tentative evidence that simple audiotape replay can be beneficial (Satel and Sledge, 1989).

Another avenue may be to provide patients with conversational opportunities (e.g. in the context of a therapeutic community). Along with the results of the present study, there are several clues to suggest that this approach might be helpful. For example, St-Hilaire and Docherty (2005) have reported a significant association between affective reactivity of speech in psychotic patients and difficulties relating to others and fear of social relationships. In line with these findings, Grant and Beck (2009) reported that evaluation sensitivity (i.e. dysfunctional beliefs about social acceptance) seems to play an important mediating role in FTD, possibly worsened by the individual's awareness of their communication difficulties (McGrath and Allman, 2000). Hence, in future clinical studies it may be useful to assess the

possible role of general supportive environments such as therapeutic communities in helping the thought disordered patient. There is some evidence that this community-based therapeutic milieu offers an important alternative to standard psychiatric care (Calton et al., 2008).

Alternatively, and perhaps less costly, may be to utilize social network interventions. These interventions have been proven to be effective in reducing isolation in socially withdrawn individuals (Terzian et al., 2013).

Another potential avenue may be to strengthen existing social skills training programmes by emphasizing components of conversational skills (e.g. Starting, maintaining and terminating a conversation, modelling and role-playing different conversational situations, etc.) and specific strategies to address social isolation in thought disordered patients (e.g. providing opportunities for pleasant experiences of conversation, scheduling positive social activities to counteract isolation, etc.). Social skills training programmes have a long history in the field of schizophrenia and consist of behavioral therapy principles and techniques aimed at helping patients improved their interpersonal and independent living skills (Kopelowicz et al., 2006). There is now evidence that these programmes are effective improving community functioning and social and daily living skills in patients diagnosed with schizophrenia (Kurtz and Mueser, 2008). In future studies, it would be relevant to study the impact that these interventions may have on FTD.

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## **Chapter 6. Communication deviance as an *envirottype* for psychosis: A systematic review and an exploration of potential developmental pathways**

This paper has been submitted and is currently under review as de Sousa, P., Sellwood, W. & Bentall, R. P. (under review). Communication deviance as an *envirottype* for psychosis: A systematic review and an exploration of potential developmental pathways. *Psychological Bulletin*.

### **6.1 Abstract**

**Background.** Over the last decade, there has been a renewed interest in the role of environmental risk factors in schizophrenia. Parental communication deviance (CD) has been subject to considerable research in the past but its impact on psychosis-risk and the mechanisms responsible for this impact have been neglected. Here we systematically review the field to consider the evidence on the role of CD in the risk of psychosis and thought disorder (TD), and factors that influence CD in caregivers. Finally, we consider possible developmental pathways from CD to offspring mental illness.

**Methods.** We performed a systematic search of the literature on CD in caregivers of offspring diagnosed with schizophrenia-spectrum disorders. Studies were identified that addressed different aspects of the construct.

**Results.** CD is a well-replicated finding in the caregivers of patients diagnosed with psychotic disorders and there is evidence suggesting a more specific association with offspring's TD. This association does not seem to be explained by potential confounders. Moreover, CD seems to be a stable characteristic that is independent of caregivers' expressed emotion and seems to be associated with offspring's relapse. Explanations based either on the influence of the offspring on the caregiver or purely genetic factors are unsatisfactory.

**Conclusions.** We argue that CD is a valid psychological construct and suggest potential developmental pathways and mechanisms by which CD may impact on the developing offspring via early episodes of joint attention (JA). We also discuss important issues that need to be addressed in future studies to move the field further.

“Developmentally, the ways in which significant persons in the child’s formative years communicate and share attention and meaning with the child are critical in the

shaping of his own patterns of thinking, communicating and experiencing” (Wynne, et al., 1977, p. 260).

## 6.2 Introduction

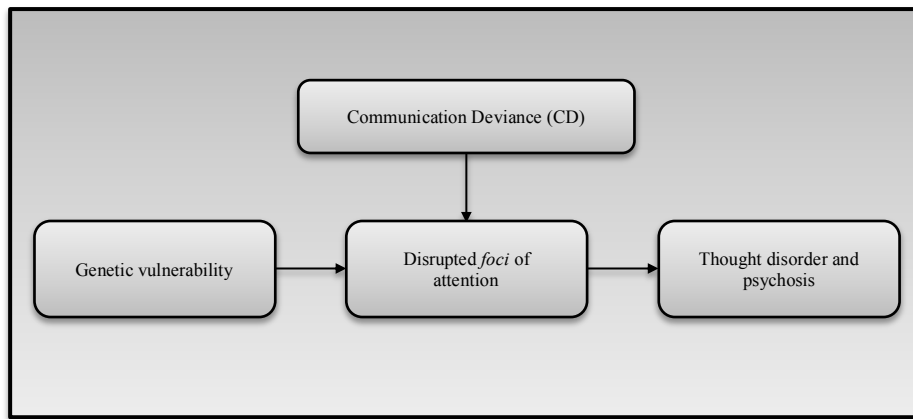
The role of upbringing in severe mental illness has long been a matter of controversy. In the middle decades of the last century some theorists argued that psychotic patients were victims of ‘schizophrenogenic’ mothers (Fromm-Reichmann, 1948), family scapegoating (Laing & Esterson, 1964), ‘skewed’ and conflictive family relations (Lidz, Cornelison, Terry, & Fleck, 1958) or double-bind communication patterns (Bateson, Jackson, Haley, & Weakland, 1956). These theories were largely discredited for several reasons. First, they were considered inconsistent with the dominant neurobiological paradigm in psychiatry (Read, Bentall, & Fosse, 2009); second, because high heritability estimates for the psychotic disorders (Sullivan, Kendler, & Neale, 2003) were sometimes mistakenly taken to mean that environmental influences were negligible (Bentall, 2009); and third, because they were considered to be highly stigmatizing, not only of patients but also of their carers (Read, Seymour, & Mosher, 2004).

In subsequent years, however, two lines of research did reveal evidence of the role of the family environment, although in ways which were more nuanced than those of the earlier accounts. On the one hand, following seminal work by the sociologist George Brown and his colleagues (Brown, Wing, Carstairs, & Monck, 1962), it became clear that a style of *reacting to* psychotic illness, known as expressed emotion (EE) and characterized by high levels of criticism, hostility and over-protectiveness or intrusiveness in the caregivers, is an important contributor to relapse (with a meta-analysis reporting significant effect-sizes for the association between EE and relapse in schizophrenia,  $r = 0.31$ ; 95% CI [0.23, 0.37], Butzlaff & Hooley, 1998).

The second line of research followed from the early work of two US researchers, Lyman Wynne and Margaret Singer, and focused on abnormal patterns of communication in families of psychotic offspring. This work on what became known as *communication deviance* (CD; Singer & Wynne, 1963, 1965a, 1965b; Wynne, et al., 1958; Wynne & Singer, 1963a, 1963b) has subsequently been supported and developed in a substantial body of studies; and yet, the potential of this work to cast a light on the developmental origins of psychosis is not widely appreciated. Here we review existing research on CD, and discuss some points of contact between CD research and research in developmental psychopathology.

### **6.2.1 Communication deviance (CD)**

Wynne and Singer's work grew out of the observation that parents of psychotic offspring would often speak in ways that compromised the development of shared meaning during communication (i.e. meanings cannot be consensually or visually validated by the listener, Singer, Wynne, & Toohey, 1978). They suggested that subtle atypicalities at the level of the establishment and maintenance of shared *foci* of attention between the parent and the child were of crucial importance in understanding the ontogeny of, and vulnerability to psychosis (see Figure 4; Singer, 1967; Singer & Wynne, 1966b; Singer, Wynne, & Toohey, 1978; Wynne, 1967; Wynne, et al., 1977). CD was conceptualized as a pattern of communication that impaired the normal flow of dialogue by rendering its content vague, contradictory and ambiguous and therefore compromising conversational alignment (Singer & Wynne, 1966b). Wynne and Singer were not interested in the proximal effect of CD but rather in its impact on the offspring's socio-cognitive development through its potential pervasive influence over time (Wynne, et al., 1977).



**Figure 4 - Wynne's (1981) CD model.**

The developmental principle is that children *learn* to share and sustain foci of attention, and consequently derive meaning from the world around them through communication with their parents, and that parents therefore have a primary role in organising the experience of the developing offspring (Wynne, 1981, 1984). This process corresponds with what today is referred to as *joint attention* (Carpenter, Nagell, & Tomasello, 1998; Mundy & Newell, 2007; Tomasello, Carpenter, Call, Behne, & Moll, 2005) or *joint engagement* (Adamson, Bakeman, & Deckner, 2004; Adamson, Bakeman, Deckner, & Nelson, 2014; Adamson & Bakeman, 2006; Bakeman & Adamson, 1984). These atypical familial *transactions*<sup>ix</sup>, in which thought and language are embedded, were hypothesized to interact with genetic vulnerability during sensitive developmental periods, and with other psychosocial stressors, leading to the escalation of psychopathology (Singer, 1967; Singer & Wynne, 1965a, 1965b; Wynne, 1967; Wynne, et al., 1977; Wynne, 1981, 1984; Wynne & Singer, 1963a, 1963b).

<sup>ix</sup> The term *transaction* was used by Wynne to emphasize the hypothesized internal change that occurred during an interaction between parent and child.

Rorschach	Thematic Apperception Test
<i>Closure problems</i>	
Uncorrected speech fragments Unintelligible remarks Unstable percepts Gross indefiniteness and tentativeness Responses in negative form Subjunctive "if" responses "Question" response Contradictory information Inconsistent and ambiguous references Incompatible alternatives	Fragments of words, phrases, and ideas appear in the story Passages of the story are unintelligible Part of the story is given as a question, or the listener is called upon to supply meaning The story is left hanging Spontaneous mention of a major perceptual element of the picture is not made (e.g. the violin on card 1) The subject is grossly uncertain about a major perceptual element. Contradictions and inconsistencies are present Responses in negative form* Responses in subjunctive form* No integration of picture elements* A card 2 figure is left out of story* Attribution of intention to the cards* "I hope" endings* "I don't know" endings* Other closure problems*
<i>Disqualifications</i>	
Derogatory, disparaging, critical remarks Nihilistic remarks Failures to verify own responses Retractions and denials Forgetting responses Partial disqualifications	
<i>Disruptive behaviour</i>	
Interruptions of examiner's speech Extraneous questions and remarks Odd, tangential, inappropriate remarks Non-verbal, disruptive behaviour Humour Swearing Hopping around among responses	Interruption of the task Disruptions of the set Questions about the task instructions after the story is begun* Associations about self which are not a part of the story* Tangential replies to examiner questions*

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\* Items introduced by Jones (1977) are identified with an asterisk.

Negativistic, temporary card rejection followed by a response  
Concrete-set responses  
References to “they” and to the intent of others

*Peculiar language and logic*

*Peculiar word usages, constructions and pronunciations*

Ordinary words or phrases used oddly or out of context  
Odd sentence construction  
Quaint, private terms or phrases  
Euphemisms  
Slips of tongue  
Mispronounced words  
Foreign terms used for no particular reason  
Cryptic remarks  
Clang associations, rhymed phrases and wordplay  
Abstract, global terms

The card is misperceived  
Idiosyncratic meaning is assigned to details  
Odd phrasing and word usage are present  
Slips of tongue occur  
Peculiar reasoning of any kind is present  
Repetitiveness of words, phrases or ideas occur  
Incorrect abstract word usage\*

*Reiteration*

Repetition of words or phrases

*Peculiar logic*

Illogical combinations of percepts and categories  
Non-sequitur reasoning  
Assigning meaning illogically on the basis of non-essential attributes of the cards  
Contaminations

*Word count: Initial viewing, inquiry, and total*

*Additional formal characteristics*

Reaction time average  
Story length index

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**Table 15** - Communication deviance (CD) categories for Rorschach and TAT protocols (Jones, 1977; Singer & Wynne, 1966b; Wynne, Singer, Bartko, & Toohey, 1977).



Examples of CD would range from the use of *ambiguous linguistic references* e.g. “Kid stuff that's one thing but something else is different too” (Velligan, et al., 1990, p. 5) and *peculiar phrases* e.g. “this man is in the process in thinking of the process of being a doctor” (Miklowitz et al., 1991, p. 166) to *nonsequitur replies* e.g. “(interviewer: So, you are an animal lover.) Oh, yeah. And my husband, who's a Capricorn and I'm a Capricorn and we stink together” (Docherty, 1993, p. 753) together with more overarching non-verbal disturbances at the level of the pragmatics of communication such as mistimed turn-taking. Table 15 shows the classification of the components of CD originally developed by the authors for the purpose of coding speech generated as parents completed projective tests such as the Rorschach (Rorschach, 1921) and Thematic Apperception Task (TAT, Murray, 1943)<sup>10</sup>. However, across the years, CD had been measured in various ways, and Table 16 describes some of the methods used by various research groups.

More recently, Docherty and colleagues (Docherty, Miller, & Lewis, 1997; Docherty, 1996b) have developed a scoring system (the communication disturbances index, or CDI) to assess discourse cohesion in communication by targeting performance on *deictic* references (following the work of Halliday & Hasan, 1976; Rochester & Martin, 1979). Deictic references are words that, despite their fixed semantic meaning, depend on the context of communication (e.g. space/location and time) for their denotational meaning (e.g. in the following sentence: “He enjoys living in this city”, the word “he” and “this” are both deictic as they index the person and the place to which the speaker refers to, respectively, and are dependent on the context of both speaker and addressee). Difficulties at the level of deixis have been noted in the children of parents diagnosed with schizophrenia (Harvey, Weintraub, & Neale, 1982)

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<sup>10</sup> Both TAT and Rorschach are projective techniques that were developed to assess personality by asking participants to elaborate a narrative around ambiguous stimuli (e.g. inkblot or a picture of people interacting). In the context of CD research, these methods were used to elicit speech samples that could be later coded.

and non-affected siblings of patients diagnosed with schizophrenia (Docherty, Gordinier, Hall, & Dombrowski, 2004) and have been reported to be associated with thought disorder (TD) and poor ‘theory of mind’ (ToM; the ability to infer the mental states of others) in patients diagnosed with schizophrenia (Docherty, 2005; Docherty et al., 2013), leading some authors to suggest that TD is associated with specific difficulties integrating contextual information (Hardy-Baylé, Sarfati, & Passerieux, 2003; Patniyot, 2011).

Despite this progress, work in this area has dwindled in the past decade and very little effort has been directed towards understanding how the observations made by investigators in psychiatry might be accounted for in terms of plausible explanatory developmental mechanisms.

Typically, findings on CD have been interpreted as evidence of an *endophenotype* of schizophrenia (Levy et al., 2010), where endophenotype refers to a distal, quantitative trait associated with the complex disorder, which is assumed to be under strong genetic control and believed to be more amenable to research due to its higher penetrance and lower genetic complexity (Gottesman & Gould, 2003). However, there has been very little evidence to support this hypothesis from molecular genetics (in fact, the only suggestive evidence comes from the familial aggregation of TD and CD, which we discuss later). An alternative approach is to consider whether parental CD (whether or not it has genetic determinants) constitutes an important aspect of the child’s social environment, which could explain its association with the emergence of psychotic symptoms during development.

The idea that social-contextual family factors may confer non-genetic vulnerability to offspring is certainly not new and has received some attention in high-risk studies of mothers diagnosed with schizophrenia (Wan, Abel, & Green, 2008) as

well as in longitudinal studies documenting an interaction between genetic risk and dysfunctional family environments in the long term prediction of psychotic-spectrum disorders (Carter, Schulsinger, Parnas, Cannon, & Mednick, 2002; Tienari et al., 2004; Wynne et al., 2006). Conversely this idea is also reflected in studies that have documented the protective effect of positive family environment in populations at risk of psychosis (González-Pinto et al., 2011).

Developmental science has progressed considerably in the years since the development of the CD concept by psychopathologists, offering rich explanatory constructs and complex pathways to explain the multiple factors at play during child development. Constructs such as *joint attention* and *shared intentionality* (Tomasello, Carpenter, Call, Behne, & Moll, 2005), maternal *mind-mindedness* (Meins et al., 2002), maternal *reminiscing style* (Fivush, Haden, & Reese, 2006), parents reflective capacity (Fonagy, Gergely, & Target, 2007) or disrupted mother-infant communication (Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009) have all been used to emphasize the crucial role of communication and engagement between the caregiver and the infant in early socio-cognitive and affective development. However, few attempts have been made to explicate how these constructs might contribute to our understanding of the impact of CD.

Methodology	Example	Description	Coding protocol
Rorschach test (Rorschach, 1921)	Hirsch & Leff (1971)	Parent is presented with a series of cards with inkblots (one at the time) and asked to tell the experimenter everything that each inkblot looks like or reminds the parent of. After this initial phase (initial viewing), the experimenter returns to the cards reminding the parent of their initial answers and asking follow-up questions (inquiry).	Transcripts are coded using Wynne and Singer's scheme for Rorschach protocols (see Table 15).
Object Sorting Test (OST; Lovibond, 1954)	Wild, Singer, Rosman, Ricci, & Lidz (1965)	In the first part of the test, parents are presented with seven familiar objects (e.g. smoking equipment, silverware, etc.) one at the time and asked to place it with the objects it belongs with. Parents are then asked to explain why the objects belong together. In the second part, parents are presented with twelve sequential sets of objects assembled around a common characteristic (e.g. all cutlery). For each set, parents are asked why the objects in the set belong together.	Verbatim transcripts are coded with codes derived from Wynne and Singer's work which include categories such as: <i>inability to maintain a consistent task set</i> (e.g. <i>fragmentation of attention, inability to maintain the role of the subject being tested</i> ), <i>blurring of meaning</i> or <i>peculiarities</i> (e.g. <i>peculiar verbalisations and imprecise referents</i> ).
Thematic Apperception Test (TAT, Murray, 1943)	Jones (1977)	Parents are invited to elaborate a story based on 7 TAT cards that are likely to elicit interpersonal and family themes.	Transcripts are coded using Wynne and Singer's scheme for TAT protocols (see Table 15).
Descriptive task	Sass, Gunderson, Singer, & Wynne (1984)	Parents are invited to explain a term of the American culture (e.g. Christmas, television, cowboys, baseball), as if they were talking	Transcripts are coded using codes derived from Wynne and Singer's work (J. Jones, 1977; Singer & Wynne, 1966b).

		to a member of a foreign culture who, though fluent in English, knows nothing about it.	
Object Relations Technique (ORT, Phillipson, 1955)	Solana (1988)	Mother and father are presented with two cards of the Phillipson's test and asked to prepare a story together for a maximum period of 20 minutes. They are informed that story needs to include past, present and future.	Transcripts are scored with 17 items derived from Wynne and colleagues' work (1977).
Family Consensus Rorschach (CR; Loveland, Wynne, & Singer, 1963)	Tompson, Asarnow, Hamilton, Newell, & Goldstein (1997)	Family is taken to an observation room equipped with video cameras and microphones and instructed to discuss for seven minutes what an inkblot on a Rorschach card (e.g. VIII) looks like, resembles or reminds them of. After this initial part, the experimenter returns to the room to ask more questions about the family's answers (inquiry).	Transcripts of the conversation are coded using three categories: <i>attentional drift</i> , <i>clarity of communication</i> , and <i>thought disorder</i> .
Problems solving	Velligan, Goldstein, Nuechterlein, Miklowitz, & Ranlett (1990)	Family (mother, father and offspring) is invited to discuss two issues identified by them as areas of conflict for approximately 10 minutes. During the interaction, they are asked to share their feelings and work towards as resolution.	Transcripts are scored using coding scheme derived from Wynne and Singer's work called Interactional Communication Deviance (ICD; Velligan et al., 1990) which comprises 7 categories: <i>idea fragments</i> , <i>unintelligible remarks</i> , <i>contradictions/retractions</i> , <i>ambiguous referents</i> , <i>extraneous remarks</i> , <i>tangential or inappropriate responses</i> , <i>odd word usage/odd sentence construction</i> .

Family interaction	Bayer (1996)	Family (mother, father and offspring) is invited to discuss offspring's personal characteristics and qualities and how he or she got to be that way for 15 minutes.	Transcripts of the conversation are coded for: <i>Egocentric utterances, disconfirmatory feedback and relationship defining communication.</i>
Communication Conflict Situation (CCS)	Rund (1986)	Parental couple is provided with two maps and one parent is instructed to direct the other to a predetermined location via a special route. On one of the maps a simple and a complicated route are marked whereas on the other these routes are unmarked and an extra street is added to the complicated route (conflict situation).	The transcripts are scored along 7 different categories: <i>peculiar language, irrelevant or disruptive behaviour, unstable perception and thinking, egocentrism, decentration, contract proposals, and attribution of communication failures.</i>
Colour Conflict Method (CCM)	Holte & Wichstrøm (1990)	Mother, father and offspring are each presented with a book containing 17 15x20 cm coloured solid plates. Families are instructed to discuss the colour they saw on each page and reach an agreement. The colours are manipulated by varying the degree of colour saturation, making agreement difficult and increasing the potential for conflict. No time limit.	Transcripts of the interaction are coded with the Confirmation-Disconfirmation Coding System (CONDIS) which includes: <i>confirmatory feedback reaction (CON); confirmation with agreement (CON-A); confirmation with neutrality (CON-N); confirmation with disagreement (CON-D); and disconfirmatory feedback reaction (DIS): egocentric utterance (EU); linear self-disqualification (LSD); paradoxical self-disqualification (PSD); and active disqualification of others (AD).</i>
Autobiographical memories	Docherty, Miller, & Lewis (1997)	Parents are invited to provide 10-minute conversational speech samples on 'good memories of pleasant non-stressful times', neutral topics such as interests and daily	Transcripts are coded with the Communication Disturbances Index (CDI, Docherty, 1996) which has 7 different categories: <i>vague references, confused references, missing information references, ambiguous word</i>

		activities and of ‘bad memories of stressful times’. The aim is to collect speech samples with different degrees of emotional salience.	<i>meanings, wrong word references, and structural unclarities.</i>
Five minute speech sample (FMSS, Magaña et al., 1986)	Kymalainen, Weisman, Rosales, & Armesto (2006)	Parents are instructed to speak about their offspring for 5 minutes, uninterrupted, describing what kind of person he or she is and how the two of them get along together.	Transcripts are coded using ICD codes (see above).
Camberwell Family Interview (CFI, Leff & Vaughn, 1985)	Cole, Kane, Zastowny, Grolnick, & Lehman (1993)	Parents are administered a semi-structured interview covering topics such as the onset of the offspring’s disorder and the symptoms that were noticeable to the parent in the period preceding an admission to an inpatient ward or a psychotic relapse. Other topics include the offspring’s participation in the household tasks and the level of expressed emotion between the family relatives. The original protocol is very long (1 ½ hour) but only the initial part of the interview is coded for CD.	CD is rated using four levels of severity: (0) absent, (1) mild, (2) moderate, and (3) severe.

**Table 16** - Some of the methodologies used to assess CD and related constructs.

## 6.3 Aims and objectives

In this systematic review, we will consider the consistency of the findings from CD research and make the case that CD can be conceptualized as a part of the *envirome* of psychosis. By *envirome*, we mean a set of multifactorial environmental circumstances (both current and in earlier life) that contribute to the onset of, or influence the course of, mental health disorders, both independently of or in combination with genetic vulnerability (Anthony, 2001; Anthony, Eaton, & Henderson, 1995). More specifically, we argue that CD constitutes a micro-social and enduring risk factor (or *envirotypes*) that increases the probability of TD in the offspring and that the observed associations between CD and psychosis are mediated by the effects of CD on the child's cognitive development.

In making this case, we will focus on six questions that have often been considered in the CD literature:

- 1) *Is CD in the caregivers of patients with psychosis a replicable and specific phenomenon?*
- 2) *Is CD in caregivers associated with thought disorder (TD) in the offspring?*
- 3) *Is CD in caregivers associated with relapse in the offspring?*
- 4) *Is CD associated with other caregiver characteristics such as expressed emotion or psychopathology?*
- 5) *Can the association between CD in the caregiver and offspring psychopathology be explained by reverse-causality (the caregivers' reactions to their disturbed children)?*
- 6) *Can the association between CD in the caregiver and offspring psychopathology be explained by genetic factors?*



After reviewing this evidence, we will consider possible developmental pathways from CD in caregivers to the development of psychopathology in their offspring.

## **6.4 Method**

### **6.4.1 Search strategy**

We searched PsycINFO for studies published between January 1959 and January 2012. The starting date of our search was the year of the first publication that examined thought disturbances in the parents of psychotic offspring (McConaghy, 1959). We used following terms: *communication deviance*, *communication disturbance\** and *thought dis\** combined with the terms *famil\**, *parent\**, *mother*, *father* combined with *schiz\** and *psycho\** using Boolean operator “and” and “or”.

### **6.4.2 Exclusion criteria**

Excluded were studies that employed “artificial family” designs (Liem, 1974; Waxler, 1974) in which the family members of psychotic offspring interact with healthy offspring and parents of healthy offspring interact with psychotic offspring. These studies raised important issues regarding ecological validity. An exception is that they will be discussed in the section about on reverse-causality, given that they are cited in discussions of this question.

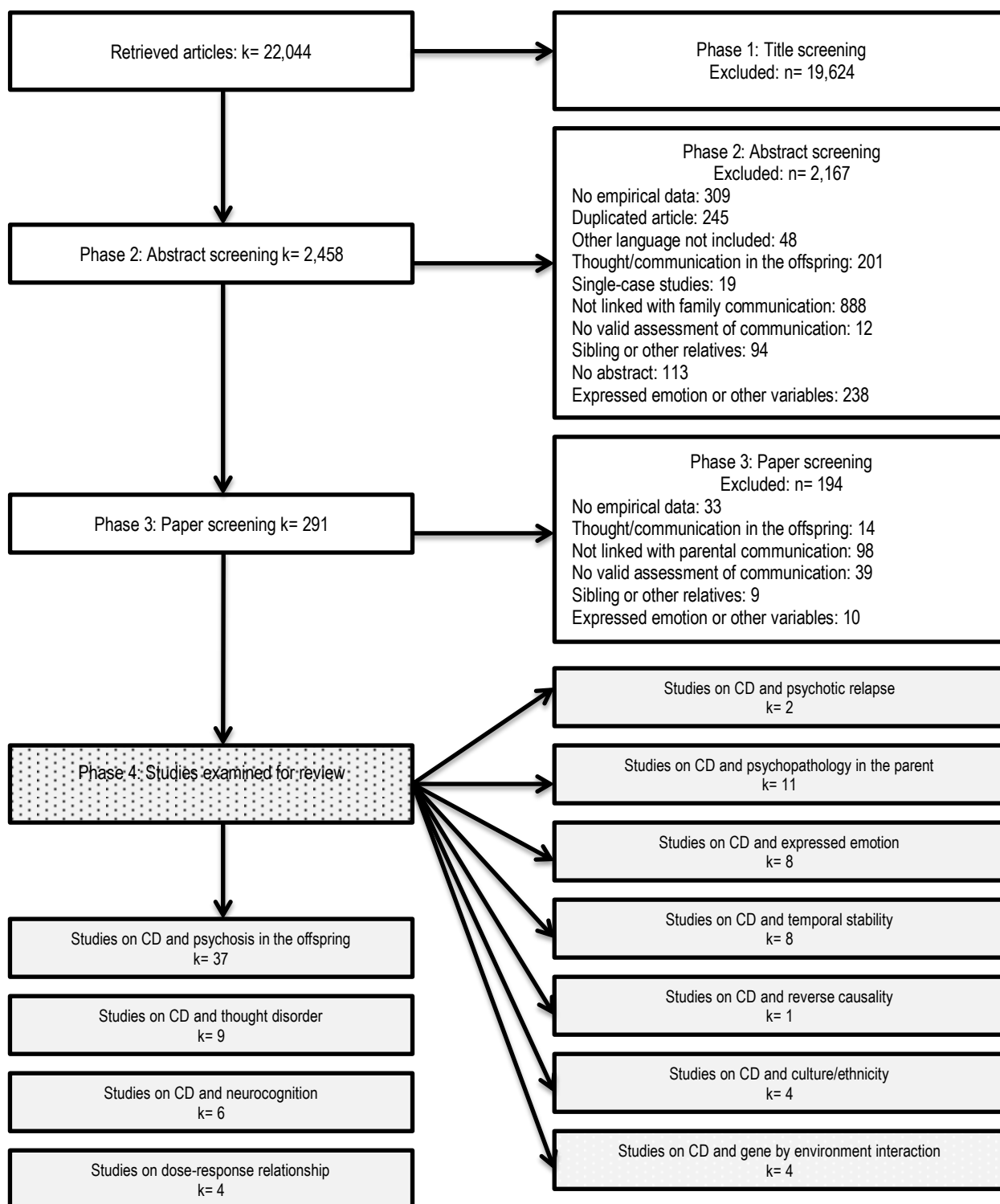
Studies where the focus was on factors potentially related to CD, but did not evaluate it were excluded. This included studies that examined concept-formation in parents of psychotic of offspring (Catts, Mcconaghy, Armstrong, Ward, & Fox, 1992; Lidz, Wild, Schafer, Rosman, & Fleck, 1962; McConaghy, 1959; Romney, 1969;

Rosman, Wild, Ricci, Fleck, & Lidz, 1964; Schopler & Loftin, 1969a, 1969b). Studies that have used consensus tasks (Loveland, Wynne, & Singer, 1963; Loveland, 1967; Singer, 1968) have also been excluded as these primarily concern problem-solving within the family, *e.g.* the family's ability to reach a consensus regarding Rorschach inkblot.

Our final exclusion criterion was studies that have examined subclinical thought disorder (TD) in the parents of psychotic offspring. Although CD and TD certainly share some common features they are not necessarily correlated (Johnston & Holzman, 1979). The same applies to studies that have primarily concerned other related concepts such as acknowledgement and disconfirmatory feedback (Holte & Wichstrøm, 1990a, 1990b; Wichstrøm & Holte, 1991, 1992) and *double-binds* (Bateson et al., 1956; Bugental, Love, Kaswan, & April, 1971). Regarding the former they will only be considered in the section on reverse-causality as they provide important clues regarding this question.

## **6.5 Findings**

Our results were cross-referenced with the search results of a larger systematic search on early experience and psychosis (Varese et al., 2012). The different phases of our search have been documented elsewhere (de Sousa, Varese, Sellwood, & Bentall, 2013) but can be seen in Figure 5.



**Figure 5** - Flowchart of the studies included in the qualitative review.

### **6.5.1 Is CD in caregivers of patients with psychosis a replicable and specific phenomenon?**

Table 17 summarizes studies published since 1963 comparing parents of offspring diagnosed with psychotic-spectrum disorders and controls on CD. Several important issues need to be highlighted when we consider the early studies carried out by Wynne, Singer and colleagues. First, these studies were marked by a noticeable diversity in the methodologies employed to elicit speech from parents and by the scoring schemes used to quantify CD. For example, some studies used data gathered from multiple psychological tests (Singer & Wynne, 1963, 1965b) whereas others have used excerpts of family therapy sessions (Morris & Wynne, 1965; Palombo, Merrifield, Weigert, Morris, & Wynne, 1967), transcripts from the object sorting task (Harrow & Quinlan, 1985; Wild et al., 1965), individual Rorschach protocols (Hirsch & Leff, 1971; Johnston & Holzman, 1979; Singer & Wynne, 1966a; Wender, Rosenthal, Rainer, Greenhill, & Sarlin, 1977; Wynne, Singer, Bartko, & Toohey, 1977), interpretation of proverbs (Wynne et al., 1977), transcripts of the consensus Rorschach (Behrens, Rosenthal, & Chodoff, 1968), individual TAT protocols (Jones, 1977), descriptive tasks (Sass et al., 1984) or transcripts from the 20 questions task (Wild, Shapiro, & Goldenberg, 1975). Moreover, in some of these studies the speech samples or the test data were used to make blind predictions about the diagnostic category or severity of the offspring's psychopathology and not to quantitatively measure CD (Morris & Wynne, 1965; Singer & Wynne, 1965b) and crucial methodological aspects such as inter-rater reliability were either not considered or at least not included in the publication (see Table 17).

Despite these important caveats, the most striking observation emerging from these studies is that nearly all have replicated Wynne and Singer's early findings,

pointing to a significantly elevated prevalence of CD in the parents of psychotic offspring when compared with both the parents of healthy controls and with the parents of offspring diagnosed with non-psychotic disorders.

One study deserves particular attention because it appears to be an exception. In an attempt to replicate these findings in the UK, Hirsch and Leff (1971, 1975) tested groups of parents of offspring diagnosed with schizophrenia and common mental health disorders using individual Rorschach protocols and Wynne and Singer's published scoring system (1966b). Although an initial analysis revealed that parents of offspring diagnosed with schizophrenia displayed significantly more CD than parents of offspring diagnosed with non-psychotic disorders ( $p < 0.05$ ), the effect disappeared when word count (verbosity) was accounted for. This finding led some researchers to believe that CD was an artefact of word count and this may have been responsible for a lack of interest in CD amongst UK schizophrenia researchers since. However, all the CD studies published subsequently have demonstrated that, when word count is accounted for in the statistical analysis or when CD is adjusted for word count or lines of speech, effect-sizes remain statistically significant and robust (e.g. Docherty, 1995; Solana, 1988; Wynne et al., 1977). For example, Johnston and Holzman (1979) also reported a significant correlation between CD scores and word count but when they adjusted CD scores for word count, the difference between the parental groups remained statistically significant, with mothers of offspring diagnosed with schizophrenia scoring higher than mothers of healthy offspring.

Another limitation of the early studies is that they have relied heavily on projective techniques to elicit speech from the parents and it could be argued that these methodologies poorly represent natural speech. Docherty and her colleagues partly overcame this problem by using speech of parents describing autobiographical

memories. In the first study, Docherty (1993) coded the verbatim transcripts of samples of speech from parents of offspring diagnosed with schizophrenia and from parents of healthy controls using an adapted version of a CD scoring system (ICD; Velligan, 1985). Consistent with previous findings, parents of psychotic offspring displayed significantly more CD than comparisons. Subsequently, Docherty (1995b; 1994) reported the same significant differences when they measured the frequency of *unclear linguistic referents* per clause of speech, with the parents of psychotic offspring displaying significantly more *unclear references* than parents of healthy controls. These findings were later replicated using the CDI (Docherty, 1996) and larger samples (Docherty, Miller & Lewis, 1997; Docherty, Hall & Gordinier, 1998; Docherty & Gordinier, 1999; Docherty, et al., 1999). These studies are important for three reasons. First, they replicated previous findings gained with projective tests using a rigorous methodology (with word count and parent's education accounted for) and gold standard diagnostic criteria (e.g. DSM-III-R, APA, 1987). Second, the quality of the parent's communication was measured in samples of more naturalistic speech; and third, they emphasize the role of poor discourse deixis as being central to the parents' communicational problems.

Source	Study type	Parents (n)	Comparisons (n)	Control groups	Education (Y/N)	≥DSM-III (Y/N)	Age of the offspring (>15)	Methodology	Scoring	Parent	IRR (Y/N)	Verbosity (Y/N)	Main findings
Singer and Wynne (1963)	CCS	40	80	Other	N	N	N	Projective	CD	Both	N	N	95% of parental couples of Scz had Rorschach protocols marked with <i>indefinite referents, perseverations, pointless and fragmented speech</i> in contrast with 60% of the parents of children who were socially withdrawn, 20% of the parents of children diagnosed as autistic and 0% of the parents of children with behavioural problems.
Singer and Wynne (1965)	CCS	40	32	Mixed	N	N	Y	Projective	CD	Both	N/a	N	Singer was able to correctly identify 85% of the parents of Scz using categories of <i>amorphous, constricted and fragmented</i> styles of communication. The predictions achieved a high level of statistical significance ( $p < 0.001$ ).
Morris and Wynne (1965)	CCS	8	8	Other	N	N	Y	Other	CD	Both	N/a	N	Parents of Scz could be significantly and blindly predicted from excerpts of family therapy sessions.
Wild et al. (1965)	CCS	44	49	Healthy	Y	N	Y	OST	CD	Both	$r = .91$	N	Parents of Scz displayed significantly more CD than parents of healthy controls. 75% of the parents of Scz had CD scores above the overall median as opposed to only 31% of the parents in the control group ( $X^2(1) = 16.55$ ,

													<p><math>p &lt; .0005</math>). Furthermore, 58% of the Scz offspring had two parents above median against 12% of controls (<math>\chi^2(1) = 8.43</math>, <math>p &lt; .02</math>). Parental groups were carefully matched for age and education,</p>
Singer & Wynne (1966)	CCS	38	80	Mixed	N	N	N/a	Projective	CD	Both	N	N	<p>Parents of Scz scored significantly higher than parents of offspring diagnosed with "neurotic" disorders and parents of healthy offspring. 32.5% of the parents of both "neurotics" and healthy controls actually scored higher than the lowest scoring parent of the Scz group and the percentage dropped to 15% when the scores were analysed by parental couple. Differentiation of parental groups was highly significant (<math>\chi^2(2) = 38.52</math>, <math>p &lt; .0001</math>). CD scores were adjusted by number of Rorschach responses.</p>
Behrens, Rosenthal, and Chodoff (1968)	CCS	56	22	Healthy	Y	N	Y	Projective	CD	Both	Y	N	<p>There were no significant differences between parents of Black and White Scz on CD, both groups scored significantly higher than parents of healthy controls. A blind rater was able to identify 85% of the families using the transcripts alone.</p>
Hirsch and Leff (1971)	CCS	40	40	Other	Y	N	Y	Projective	CD	Both	$r = .87$	Y	<p>Parents of Scz displayed significantly more CD than parents of patients with non-psychotic disorders (<math>t(78) = 2.22</math>, <math>p &lt; 0.05</math>) however this</p>



													effect disappeared when word count was partialled out.
Wild, Shapiro and Goldenberg (1975)	CCS	72	102	Mixed	Y	N	Y	Other	CD	Both	Y	Y	It was the combination of the mothers' <i>amorphous attention</i> scores and fathers' <i>closure problems</i> ( $X^2= 9.99, p< .01$ ) that proved to be the most powerful discriminator between the parents of Scz, parents of hospital controls and parents of healthy controls.
Wynne, Singer and Toohey (1976)	CCS	35	20	Other	N/K	N	Y	Projective	CD	Both	N	Y	Both adoptive and biological parents of Scz displayed significantly more CD than adoptive parents of non-psychotic offspring even after adjusting for word count ( $p< .001$ ) but there were no significant differences between biological and adoptive parents of Scz. The same differences were found when authors analysed the scores of fathers and mothers individually.
Wynne et al. (1977)	CCS	88	140	Mixed	Y	N	Y	Projective	CD	Both	$ICC= .96$	Y	Parents of Scz displayed significantly more CD than any of the three groups of comparisons even after controlling for SES, age, sex of the offspring, parents' psychopathology, years of education and word count. Parents of unremitted Scz patients displayed the highest scores. CD in mother and father significantly explained severity of offspring's psychopathology ( $F= 25.22, p< .0005$ ; $F= 27.30, p< .0005$ , respectively).

Jones (1977)	CCS	15	12	Mixed	Y	N	Y	Projective	CD	Both	$r = .86$ ( $p < .0005$ )	Y	Scores on <i>misperceptions</i> and closure problems significantly discriminated parents of hospitalized Scz.
Wender et al. (1977)	CCS	56	28	Other	N	N	Y	Projective	CD	Both	$r = .56$	Y	Biological parents of Scz displayed significantly more CD than both adoptive parents of Scz and parents of patients with LD ( $\chi^2(1) = 4.33$ , $p < .05$ ). Biological mothers of Scz had significantly higher CD scores than adoptive mothers of Scz and biological mothers of LD patients ( $\chi^2(1) = 7.92$ , $p < .005$ ). Differences amongst fathers in the three groups were non-significant.
Goldstein, Rodnick, Jones, McPherson, & West (1978)	PCS	32	N/a	Mixed	N	Y	Y	Projective	CD	Both	Y	Y	At five-year follow-up, 86% (6/7) of schizophrenia-spectrum cases had parents with high CD on the TAT as opposed to 78% (7/9) of non-spectrum cases that had parents with intermediate or low CD ( $p = .025$ ).
Johnston and Holzman (1979)	CCS	24	34	Mixed	Y	N	Y	Projective	CD	Both	Y	Y	Parents of Scz displayed significantly more CD than parents of healthy controls and non-psychotic offspring ( $p < .01$ ). These differences were only significant for mothers there were no significant differences between fathers' scores across groups.
Doane, West, Goldstein, Rodnick, & Jones (1981)	PCS	74	N/a	Mixed	N	Y	Y	Projective	CD	Both	Y	Y	At five year follow up, 9.5% of the offspring with parents with low to intermediate levels of

													CD on the TAT had been diagnosed with schizophrenia-spectrum disorder as opposed to 56% of the offspring of parents who displayed high CD. There was a significant relationship between parental CD and psychotic outcome ( $X^2(2) = 10.3, p < .01$ ). The combination of high CD and negative affective style was a significant predictor of psychotic outcome in the offspring. There were no significant associations between CD and parent's IQ or SES.
Goldstein (1981)	PCS	80	N/a	Mixed	N	Y	Y	Projective	CD	Both	Y	Y	At five-year follow-up, only parents with high CD had an offspring who subsequently received schizophrenia-spectrum diagnosis and all spectrum outcomes occurred in families where parents expressed negative or intermediate affective style statements. High CD and negative AS profile together predicted subsequent schizophrenia-spectrum diagnoses fairly precisely.
Sass et al. (1984)	CCS	42	8	Other	Y	Y	Y	Projective	CD	Both	$r_s > .84$	Y	Parents of Scz who had high scores on thought disorder achieved significantly higher CD scores ( <i>disorganisation and impaired focus</i> ) than parents of offspring with non-Scz diagnosis on two different tasks. Parental groups were matched on age, IQ and SES.
Harrow and Quinlan (1985)	CCS	40	37	Other	Y	Y	Y	OST	CD	Both	$r = .99$	N	Mothers of Scz displayed significantly more

													<i>transactional thinking</i> disturbances during the OST than mothers of non-psychotic offspring ( $F(1,36)=4.59, p< .05$ ). There were no differences between groups of fathers.
Rund (1986)	CCS	42	58	Mixed	Y	Y	Y	Projective/CCS	CD/EU	Both	Y	Y	Parents of Scz displayed significantly more CD than both parents of non-psychotic controls and parents of healthy controls on the CCS. There were also significant differences within the Scz group with parents of non- paranoid offspring displaying significantly more CD than parents of paranoid offspring. Groups were matched on age, education and social class.
Goldstein (1987)	PCS	128	N/a	N/a	Y	Y	Y	Projective	CD	Both	Y	Y	Parental CD alone ( $X^2=17.90, p< .001$ ) and in combination with negative affective style was a significant predictor of schizophrenia-spectrum diagnoses in the offspring at 15-year follow-up.
Asarnow, Goldstein and Benmeir (1988)	CCS	28	72	Mixed	Y	Y	N	Projective	CD	Mother	$K=.77 (p< .01)$	Y	There was a significant association between the child having a schizophrenia- spectrum diagnosis and the parent displaying high CD on the TAT protocol ( $X^2(1)=6.09, p< .02$ ). This was especially the case for mothers ( $X^2(1)=8.24, p< .005$ ) with 82% (9/11) of the mothers of children diagnosed with Schizotypal traits displaying high CD. There were no between group differences in word count.
Solana (1988)	CCS	40	40	Other	Y	Y	Y	Projective	CD	Both	$r=.87$	Y	

													Parents of Scz displayed significantly more CD than parents of children with learning difficulties even after accounting for word count ( $t=3.92$ , $p<.001$ ). This was true for both mothers and fathers. Parental groups were matched on socio-demographic variables and IQ.
Tompson et al. (1990)	CCS	18	10	Other	Y	Y	N	Projective	CD	Both	70%	Y	There were no significant differences between parents of Scz and parents of children diagnosed with depressive-spectrum disorders on CD (interaction task).
Holte and Wichstrøm (1990)	CCS	14	28	Mixed	Y	Y	Y	CCM/CR	EU	Both	$ks>.80$	N	Parents of Scz displayed significantly more <i>egocentric utterances</i> and <i>active disqualifications</i> during the CCM than parents of patients with other diagnosis (both $p<.05$ ) and parents of healthy controls ( $p<.03$ ; $p<.001$ , respectively).
Miklowitz et al. (1991)	CCS	53	32	Other	Y	Y	Y	Projective/Family	CD/ICD	Both	$ICC=.79$	Y	There were no significant differences between parents of Scz and parents of patients diagnosed with Bipolar Affective Disorder (BPAD) on TAT/CD. Analysis per CD revealed that parents of BPAD scored significantly higher on <i>contorted</i> , <i>peculiar language</i> ( $p<.004$ ). In the analysis of the ICD scores (ICD) again there were no significant differences on overall scores however, parents of BPAD scored significantly higher on <i>odd</i>

													word usage ( $p < .002$ ).
Wichstrøm and Holte (1991)	CCS	34	58	Mixed	N	Y	Y	CR	EU	Both	$k = .81$	Y	Parents of Scz displayed significantly more egocentric utterances and active disqualifications than parents of patients with other diagnosis and parents of healthy controls.
Docherty (1993)	CCS	18	10	Healthy	Y	Y	Y	ABM	ICD	Both	$ICC = .95$	Y	Parents of Scz displayed significantly more CD than parents of healthy controls ( $U = 12, p = .0002$ ). Differences were significant for: <i>uncompleted</i> and <i>unintelligible remarks</i> , and <i>ambiguous referents</i> . Parental groups were matched on sex, age, ethnicity, education, occupational level, and level of functioning.
Docherty, Sledge and Wexler (1994)	CCS	18	10	Healthy	Y	Y	Y	ABM	Other	Both	$ICC = .95$	Y	Parents of Scz displayed significantly more <i>unclear linguistic references</i> per clause of speech than parents of healthy controls ( $F(1,26) = 6.85, p < .02$ ). None of the groups displayed affective reactivity of speech (worsening of communication during negative interview). Parental groups were matched on sex, age, ethnicity, education, occupational level, and level of functioning.
Docherty (1995)	CCS	18	10	Healthy	Y	Y	Y	ABM	Other	Both	$ICC = .95$	Y	Parents of Scz displayed significantly more unclear linguistic references per clause of speech than parents of healthy controls.

Bayer (1996)	CCS	40	16	Other	N	Y	Y	Family	EU	Both	$p < .001$	N	Mothers of Scz displayed significantly less <i>relationship defining</i> communication than mother of patients with other mental health diagnosis ( $F = 7.36, p < .05$ ) and fathers used significantly more <i>egocentric communication</i> than their counterparts ( $F = 10.35, p < .01$ ) during 15-minute communication about the offspring's personal qualities.
Tompson et al. (1997)	CCS	20	36	Mixed	Y	Y	N	Projective	Other	Mother	$ks > .72, (p > .001)$	Y	Mothers of Scz displayed significantly more thought disorder ( $F(1,35) = 6.39, p < .05$ ) and less communication clarity ( $F(1,35) = 4.82, p < .05$ ) than mothers of healthy controls but <i>less</i> attentional drift. There were no significant differences between Scz group and mothers of children diagnosed with depressive-spectrum disorders.
Docherty, Miller and Lewis (1997)	CCS	18	10	Healthy	Y	Y	Y	ABM	CDI	Both	$ICC = .94$	Y	Parents of Scz displayed significantly more instances of communication disturbances than parents of healthy controls when asked to described 'good, pleasant and non-stressful memories' ( $p < .001$ ). Differences were significant for: <i>vague</i> references, <i>ambiguous</i> word meanings, and <i>structural</i> unclarities. Parental groups

													<p>were matched on sex, age, ethnicity, education, occupational level, and level of functioning.</p>
Docherty, Hall and Gordinier (1998)	CCS	46	23	Healthy	Y	Y	Y	ABM	CDI	Both	ICC= .94	Y	<p>Parents of Scz displayed significantly more communication disturbances per 100 words of speech than parents of healthy controls on both the positive and negative speech samples but did not show affective reactivity of speech across conditions with the exception of <i>confused references</i> (<math>F(1,91)= 11.79</math>, <math>p&lt; .002</math>)).</p> <p>Parental groups were matched on sex, age, ethnicity, education, occupational level, and level of functioning.</p>
Docherty et al. (1998)	CCS	48	32	Healthy	Y	Y	Y	ABM	CDI	Both	ICC= .94	Y	<p>Parents of Scz displayed significantly more communication disturbances per 100 words of speech than parents of healthy controls (<math>F= 19.2</math>, <math>p&lt; .001</math>) and differences were significant for: <i>vague, confused and missing information references, ambiguous word meanings, and structural unclarities</i>. There was no association between education, occupation or level of functioning and total CDI rating.</p>
Docherty et al. (1999)	CCS	42	23	Healthy	Y	Y	Y	ABM	CDI	Both	ICC= .94	Y	<p>Parents of Scz displayed significantly more communication disturbances than parents of healthy controls (<math>F= 22.0</math>, <math>p&lt; .001</math>).</p>



													Differences were significant for: <i>vague, confused and missing information references, ambiguous word meanings, and structural unclarities</i> . Parental groups were matched on sex, age, ethnicity, education, occupational level, and level of functioning.
Docherty and Gordinier (1999)	CCS	59	24	Healthy	Y	Y	Y	ABM	CDI	Both	<i>ICC</i> = .94	Y	Parents of Scz displayed significantly more communication disturbances than parents of healthy controls. Parental groups were matched on sex, age, ethnicity, education, occupational level, and level of functioning.
Wahlberg et al. (2004)	PCS	258	N/a	Mixed	Y	Y	Y	Projective	CD	Both	<i>ICC</i> > .76	Y	In logistic regression analyses predicting psychiatric disorders in the adoptee, the main effects of genetic risk and CD in the adoptive parents were non-significant, but the GxE interaction effect was significant [OR= 10.00 95% CI (1.00–99.73), <i>p</i> = 0.05]. Amongst all adoptees (high and low risk), there were only 7 with a diagnosis of schizophrenia-spectrum disorder but 4 out of 7 (57.1%; 4/109, 3.7%) were high-risk and had adoptive parents with high CD however neither the main effects nor the interaction term were significant (underpowered).

Note: CCS, case-control study; CD, communication deviance; CDI, Cognitive Disturbances Index; DSM-III, Diagnostic and Statistical Manual of Mental Disorders-III; EU, egocentric utterances;

*healthy*, parents of healthy offspring; *ICD*, interactional communication deviance; *IRR*, inter-rater reliability reported; *mixed*, multiple contrast groups; *N*, no; *N/a*, not applicable; *N/K*, not known; *other*, parents of patients with other mental health diagnoses; *PCS*, prospective cohort study; *projective*, Rorschach, TAT, or Phillipson; *Y*, yes; *ABM*, autobiographical memories; *OST*, Object Sorting Task; *BPAD*, parents of offspring diagnosed with Bipolar Affective Disorder; *family*, family interaction; *CCM*, colour conflict method; *CR*, consensus Rorschach; *CCS*, communication conflict situation.

**Table 17** - Studies comparing parents of patients diagnosed with Schizophrenia with other parental groups.

Another important limitation of the studies considered thus far is that none coded or scored CD during family interactions. This is a very important constraint given that, for Wynne and Singer, the degree of disturbance in family transactions was hypothesized to be greater and qualitatively different from the CD found in individual family members (Wynne & Singer, 1963) as exemplified in the following excerpt provided by them:

“Daughter (presenting patient), complainingly: Nobody will listen to me.  
Everybody is trying to still me.

Mother: Nobody wants to kill you.

Father: If you’re going to associate with intellectual people, you are going to have to remember that still is a noun and not a verb.” (p. 195).

In this example, the parental replies ignore and undermine the offspring’s communication and compromise the opportunity for the generation of meaning. As the miscommunication builds up over time, the extent of this *disqualifying* family transaction can only be fully appreciated by considering the interactive context of the utterances of the different family members.

Some researchers have attempted to address this issue by coding family interactions (e.g. all the studies that have employed Velligan's ICD, Miklowitz et al., 1991; Velligan et al., 1996; Velligan, Christensen, Goldstein, & Margolin, 1988; Velligan, Funderburg, Giesecke, & Alexander, 1995; Velligan, Goldstein, Nuechterlein, Miklowitz, & Ranlett, 1990). Rund and his colleagues (1985, 1986; Rund & Blakar, 1986) tested parents of psychotic offspring and controls using the communication conflict

situation (CCS; see Table 16) and reported that they had significantly higher scores on both CD and *egocentric communication* (communicational acts that do not take into account the communicational needs of the listener, disrupting conversational alignment due to its vagueness, contradictory nature or unintelligibility). In another series of studies carried out by a Norwegian research group (Holte & Wichstrøm, 1990a, 1990b, 1991; Wichstrøm & Holte, 1991a, 1991b, 1992), families of participants diagnosed with schizophrenia and controls were assessed using the newly developed colour conflict method (CCM; see Table 16), which relies heavily on family communication during experimentally manipulated conflict and non-conflict conditions. In these circumstances, the parents of psychotic offspring produced significantly more *egocentric utterances* when they worked together. Wichstrøm and Holte (1991a) later analysed interactions between the parents (offspring were not present) during the consensus Rorschach and reported similar findings.

Finally, Bayer (1996) coded the transcripts of conversations of families of offspring diagnosed with schizophrenia and other mental health diagnoses in a situation where the whole family had been invited to discuss the offspring. The researchers reported significant group differences, with the families of psychotic offspring displaying more instances of *egocentric communication* during the conversation.

Consistent with the interactional nature of CD, some studies with parents of at-risk offspring have also reported that, during family interactions, CD is associated with less task-focused comments by parents (Lieber, 1977), less acknowledgement of the offspring (Herman & Jones, 1976) and with less facial expressiveness and eye contact by parents (Lewis, Rodnick, & Goldstein, 1981). This latter finding is potentially extremely

important as it suggests that the impact of CD may be crossmodal and not entirely contingent on the verbal content of communication.

Another limitation of the corpus of CD studies is that the large majority have employed cross-sectional case-control designs, with the evidence for CD in parents of psychotic offspring being largely gathered by measurements taken at one time-point. An exception was the UCLA high-risk study (Doane et al., 1981; Goldstein, 1981, 1987). In this prospective investigation, 64 families of young people who had been referred to a child guidance clinic because of behaviour problems were followed up for 15 years (the mean age of the offspring at follow-up was 30 years of age). CD, affective style (AS, Doane, et al., 1981) and EE assessed by the Camberwell Family Interview (CFI, Vaughn & Leff, 1976) were measured when the offspring were in early adolescence and did not display psychotic symptoms. At the 15-year follow-up it was the combination of high CD and negative AS that significantly predicted broad-spectrum schizophrenia diagnoses in the offspring (DSM-III-R, APA, 1987). Furthermore, when Goldstein's team included data from the siblings of their index children they found that a surprising 74% of parental couples with high levels of CD had at least one offspring with a broad-spectrum diagnosis of schizophrenia (although this broad spectrum category included diagnoses such as Borderline Personality Disorder). CD alone was a significant predictor of psychosis with moderate to high CD being found in the parents of 15 out of 16 offspring diagnosed with schizophrenia-spectrum disorders.

More recently, Wahlberg and colleagues (2004) studied CD as a predictor of mental health diagnoses in 109 adoptees who were part of a Finnish adoption study. CD was measured in the adoptive parents using the Rorschach and genetic risk in the adoptee

was indexed by having a biological mother diagnosed with a schizophrenia-spectrum diagnosis. At 14-year follow-up, it was the interaction between CD in the adoptive parent and genetic high-risk status in the adoptee that produced a significant odds ratio for psychiatric disorder in the offspring ( $OR= 10.00$ , 95% CI [1.00, 99.73]). It is worth noting that, at initial assessment, none of the adoptees had a diagnosable psychiatric disorder (median age 18 years old). Interestingly, examination of CD in the adoptive parent alone also yielded a very significant and robust effect-size ( $OR= 4.22$ , 95% CI [1.43, 12.46]). Unfortunately (or perhaps fortunately), there were only 7 adoptees diagnosed with schizophrenia-spectrum diagnosis (DSM-III-R, APA, 1987), making statistical analysis impossible. Nevertheless, Wahlberg and colleagues' study raises the question of whether CD is specific to parents of psychotic offspring or if it is a more general risk factor for a range of psychiatric disorders.

Some studies that have included groups of parents of offspring with non-psychotic diagnoses have documented some degree of CD in these groups (Hirsch & Leff, 1971; Johnston & Holzman, 1979; Rund & Blakar, 1986; Wynne, et al., 1977). Notably, Miklowitz and colleagues (1991) reported no significant differences on CD total scores between parents of offspring diagnosed with schizophrenia and parents of offspring diagnosed with bipolar disorder. This study used a robust methodology to assess CD including both individual protocols (TAT; Jones, 1977) and transcripts of family communication (ICD; Velligan, Goldstein, Nuechterlein, Miklowitz, & Ranlett, 1990). The only difference found was that parents in the mood disorder group displayed significantly more *odd word usage* and *contorted and peculiar language*.

A few studies have reported that parents of patients diagnosed with schizophrenia display significantly more CD than parents of offspring diagnosed with learning difficulties (LD) (e.g. Solana, 1988; Wynne, Singer, & Toohey, 1976). This finding is important given that some studies have shown that the mothers of children with LD tend to have elevated CD scores (Ditton, Green, & Singer, 1987; Rasku-Puttonen, et al., 1994) and given that there may be an association between severity of maternal CD and offspring's specific language difficulties (Poikkeus, et al., 1999).

Several other studies using comparison groups of parents of offspring with other mental health problems have provided some support for the specificity hypothesis (Bayer, 1996; Holte & Wichstrøm, 1990a; Rund, 1986; Singer & Wynne, 1966a; Wichstrøm & Holte, 1991). Moreover, of all of the studies that have either compared parents of adult offspring diagnosed with schizophrenia with parents of adult offspring with other mental health diagnosis (see studies with "other" control groups in Table 17) or with multiple parental groups (see studies with "mixed" control groups in Table 17) only a few failed to replicate Wynne and Singer's findings.

In probably the study that best addressed the issue of specificity, Wynne, Singer, Bartko and Toohey (1977) tested 228 parents using Rorschach protocols (Singer & Wynne, 1966b). The study included parents of healthy offspring, offspring diagnosed with common mental health disorders ('neurotic'), offspring diagnosed with personality disorders and two groups of parents of offspring diagnosed with schizophrenia (in one group the psychotic offspring were considered to be fully recovered and the group was designated as 'remitted', in the other the offspring had not successfully responded to treatment and were designated as 'treatment resistant'). The groups were rated on a 7-

point scale representing the severity of psychopathology (see Wynne et al., 1977 for details). As expected, parents of offspring diagnosed with schizophrenia scored significantly higher than the comparisons in the other three groups, with the parents in the ‘treatment-resistant’ group displaying the highest scores ( $2.37 \pm .14$ ) closely followed by the parents in the ‘remitted’ group ( $2.13 \pm .11$ ). Interestingly, adjusting for word count actually increased the effect-size. Furthermore, CD in both parents was reported to be the best predictor of severity of psychopathology in the offspring (CD fathers:  $F= 27.30, p< .0005$ , CD mothers:  $F= 25.22, p< .0005$ ) suggesting a dose-response relationship (in fact, the distribution of the CD scores across the five groups seemed to display a sigmoidal pattern, with high scores in the two schizophrenia groups, low scores in the healthy and neurotic groups, and the scores of the personality disorder group – which offspring patients reporting transient psychotic symptoms – falling in the middle).

This exposure-response relationship was later supported in other studies. For example, Docherty, Sledge, and Wexler (1994) reported a significant association between communication disturbances in the parent and offspring’s lifetime severity of positive and negative symptoms (PANSS, Kay & Opler, 1982). More specifically, poor linguistic performance in the more disordered parents was significantly associated with severity of auditory verbal hallucinations and delusional beliefs in the offspring. These findings were later replicated by the same group (Docherty et al., 1997) who again found a significant association between communication disturbances in parents and offspring’s lifetime severity of positive symptoms. In a third and final study, Docherty and colleagues (1999) also reported significant positive correlations between communication disturbances in parents and their offsprings’ symptom ratings on the Brief Psychiatric Rating Scale



(BPRS; Overall & Gorham, 1962). Finally, despite not primarily being concerned with the dose-response issue, Miklowitz and colleagues (1991) reported more specific associations between *contorted, peculiar language* and *odd word usage* in parents and BPRS *activation* and *hostile-suspiciousness* scores in offspring diagnosed with bipolar disorder and schizophrenia.

A few studies have investigated the prevalence of CD in the families of psychotic children. Asarnow, Goldstein and Ben-Meir (1988) tested the parents of 8-13 years olds diagnosed with psychotic- and mood-spectrum disorders using TAT protocols (Jones, 1977). They reported that it was the parents of children diagnosed with schizotypal traits that achieved the highest proportion of high-scorers (91%) followed by schizophrenia (79%), depression (53%) and dysthymia groups (50%). Tompson and colleagues (1990) assessed largely the same sample of families using the consensus Rorschach scored along three CD categories: *thought disorder*, *lack of communication clarity* and *attentional drift* (Doane & Singer, 1977). In this second study no significant differences were found between the mothers of children diagnosed with psychotic- and mood-spectrum disorders. In their final publication in the series, Tompson and colleagues (1997) used the same task but this time added a group of healthy controls. Although the differences between mothers of psychotic children and healthy controls were significant, the researchers did not find any significant differences between the mothers of children diagnosed with psychotic- and mood-spectrum disorders.

How might these findings from child samples be interpreted? On the one hand, it might be argued that they undermine the specificity of CD for psychosis. However, another possibility is that, in these studies, children who were developmentally at risk of

developing psychosis may have been over-represented in the comparison groups. Several birth cohort studies have found that withdrawal-internalizing behaviours in childhood and adolescence are significant predictors of later psychosis (Tarbox & Pogue-Geile, 2008) and non-psychotic symptoms are common in young people experiencing the prodromal phase before a first psychotic breakdown (Yung & McGorry, 1996). In this context, it is relevant that Velligan and colleagues (1988) found a significant association between mothers' CD and their school-aged children's scores on *internalizing behaviours*, *social withdrawal*, and *schizoid behaviour* as assessed using the Child Behaviour Checklist (CBC; Achenbach, 1978). In this study, none of the children were psychotic and families had been recruited because of marital or parent-child relational problems.

Taken together, the results of the studies reviewed in this section suggest a series of conclusions. First, CD is a highly replicable phenomenon, repeatedly found in the parents of patients with psychosis by independent research groups across the world using a variety of methodologies. In support of this, we recently conducted a meta-analysis addressing this question, finding a significant pooled effect-size for the prevalence of CD in parents of psychotic offspring ( $g = 0.97$ , 95% CI [0.76, 1.18]) (de Sousa et al., 2013), a finding that has recently been replicated by another independent research group (Roisko, Wahlberg, Miettunen, & Tienari, 2014). Importantly, studies have reported a dose-response relationship between CD in the parent and severity of psychotic symptoms in the offspring. This observation is important because, as famously noted by Hill (1965), when there is a consistent sizeable association between a risk factor and an outcome the presence of a dose-response relationship increases confidence that the association is causal.

A sub-analysis of the data in our own meta-analysis (Sousa et al. 2013) revealed that the effect-size for mothers ( $g = 0.89$ , 95% CI [0.54, 1.24]) was higher than the one for fathers ( $g = 0.39$ , 95% CI [0.07, 0.7]) and the difference was statistically significant ( $Q[1] = 4.38$ ,  $p < .05$ ). Two explanations are possible for this finding. First, it could represent a sex-linked inheritance effect. Second, it could represent an environmental effect caused by the child's greater exposure to the mother. Although this second hypothesis seems more intuitively likely, given the lack of research in this area both hypotheses should be considered in future research.

A second conclusion concerns the role of verbosity as a confounder. In all but one study (Hirsch & Leff, 1971), the association between parental CD and offspring psychosis survives when controlling for parental verbosity and other possible confounds such as parental educational attainment.

Third, CD is observable in family interactions and not only in less ecologically convincing test situations such as when completing the Rorschach or TAT protocols. In several studies where either the family as a whole or the parental couple have been instructed to converse about a topic, CD has been identified and the results have been statistically significant even when multiple comparison groups of parents have been employed.

Fourth, despite being significantly more prevalent in the parents of psychotic offspring, CD is not a discrete and categorical phenomenon (there is no "point of rarity" between functional and dysfunctional communication styles). It is clear from the studies reviewed that CD is also present in parents of patients with other mental health diagnoses (Wynne et al., 1977), parents of at-risk offspring (Velligan et al., 1988) and, to a lesser

degree, parents of healthy offspring (Docherty, 1993, 1995). It may well be that CD in parents of psychotic offspring may be better explained by a model where it has a particular potency for psychosis above a certain cut-off point but CD is a more general risk factor below that same cut-off point (e.g. Jones, 1977).

Finally, with regard to the specificity question, the study carried out by Miklowitz and colleagues (1991) is especially relevant, because it reported high levels of CD in the parents of offspring with bipolar disorder. In this context, it is hard to ignore increasing evidence that categorical diagnoses do not identify discrete disorders (Bentall, 2003). In particular, schizophrenia and the affective psychoses appear to overlap in terms of symptoms, course, and neurobiology (Tamminga et al., 2014), and can be encompassed within multidimensional models that include at least five dimensions (positive symptoms, negative symptoms, cognitive disorganisation, depression and mania; Demjaha et al., 2009; van Os & Kapur, 2009), possibly with an additional general psychosis dimension (Reininghaus, Priebe, & Bentall, 2012). In order to move the field forwards, future studies will need to take account of this complexity, perhaps by employing multi-diagnostic samples and considering whether any particular transdiagnostic dimensions of psychopathology in offspring are related to parental CD.

### **6.5.2 Is CD in caregivers associated with thought disorder (TD) in the offspring?**

The question of diagnostic specificity brings us to Wynne and Singer's working hypothesis that CD was specifically associated with TD in the offspring (Wynne & Singer, 1963a, 1963b), a symptom that is highly prevalent in individuals diagnosed with

schizophrenia (Roche, Creed, MacMahon, Brennan, & Clarke, 2014) but which is also found in other diagnostic groups (McKenna & Oh, 2005), particularly patients diagnosed with bipolar disorder (Andreasen & Grove, 1986; Tai, Haddock, & Bentall, 2004).

In one of their early papers, Singer had been able to blindly predict severity of offspring's TD from the parents' test data, indicating some degree of symptom specificity (Singer & Wynne, 1965b). However, not many studies have carefully considered this issue and, to the best of our knowledge, none has satisfactorily controlled for co-occurrence of symptoms when addressing this question. Controlling for co-occurrence is potentially important because, otherwise, misleading associations might be observed. For example, we have previously found that abnormal meta-cognitive beliefs, which have been theoretically linked to hallucinations (Morrison, 2001), are not specific to hallucinations when the co-occurrence of delusions and mood problems is taken into account (Varese, Barkus, & Bentall, 2011; Varese & Bentall, 2011). Similarly, insecure attachment has been theoretically associated with both hallucinations and paranoid beliefs (Berry, Barrowclough, & Wearden, 2008) but the association is only with paranoia when both symptoms are considered (Pickering, Simpson, & Bentall, 2008).

In the case of TD, this issue may be important because TD has been shown to be already present in 9 year-old children at genetic-risk for psychosis who were later diagnosed with psychotic disorders (Ott, Roberts, Rock, Allen, & Erlenmeyer-Kimling, 2002). TD has also been shown to be highly predictive of psychosis onset in adults with a high-risk mental state (prodromal symptoms) for psychosis (Bearden, Wu, Caplan, & Cannon, 2011; Demjaha, Valmaggia, Stahl, Byrne, & McGuire, 2012; DeVylder et al.,

2014). Hence, it is possible that, during the course of psychosis, TD often appears earlier than other symptoms.

In probably the best known study designed to address this issue, Sass and colleagues (1984) tested the parents of both offspring diagnosed with schizophrenia and offspring diagnosed with other psychiatric disorders, assembling the former group according to offspring's level of TD (as recorded in clinical notes). CD in the parents was assessed using individual TAT protocols and with a task designed to elicit speech (D-task, see Table 16). As predicted, parents of high TD offspring had significantly more CD on both the TAT and the D-task than parents of offspring with other psychiatric diagnoses and also than parents of offspring with a diagnosis of schizophrenia but with mild to moderate levels of TD. Curiously, the parents of the offspring with other psychiatric diagnoses actually scored higher than the parents of offspring diagnosed with schizophrenia but with mild TD. As the authors suggested, these findings may in part explain why some authors found a sizeable overlap on CD scores for parents of psychotic and non-psychotic offspring (Hirsch & Leff, 1971; Singer & Wynne, 1963). As already noted, contrary to what was once believed, TD is a transdiagnostic and non-discrete symptom that can be identified even in non-clinical populations (Weeks & James, 1995; Andreasen, 1979). Unfortunately, the evidence from this study can only be regarded as tentative given that the authors did not control for comorbid symptoms (e.g. paranoia, hallucinations, etc.).

Rund (1986) tested 50 parental couples using both the TAT and the communication conflict situation (CCS) and found that parents of schizophrenia displayed significantly more CD than comparisons. However, within the group of parents

of offspring diagnosed with schizophrenia, the parents of non-paranoid offspring displayed significantly higher CD scores than parents of paranoid offspring (means and standard deviations were, respectively:  $46.13 \pm 24.79$  and  $28.56 \pm 30.58$ ). Moreover, whilst the scores of the parents of non-paranoid offspring were significantly higher than the scores of comparisons ( $F[1, 40] = 16.41, p < .01$ ), no significant differences were found between parents of paranoid offspring and the parents of offspring with other psychiatric diagnosis and the parents of healthy controls. A high proportion of the offspring in the non-paranoid group had been diagnosed with disorganised schizophrenia, a subtype whose prominent characteristic is TD and behavioural disorganisation (*disorganisation syndrome*). Unfortunately, however, Rund did not measure TD directly.

Four studies from the Finnish adoptive family study have reported significant associations between CD in the adoptive parents and TD in adoptees. In one of the many publications that came out of this project, Wahlberg and colleagues (1997) tested if CD in the adoptive parent was a significant predictor of TD (*Index of primitive thought*, Friedman, 1952) in 154 adoptees. Interestingly, TD increased steeply in high-risk adoptees with the increase of CD in the adoptive parent. A significant odds ratio was reported for predicting the probability of TD in the adoptee from the interaction between genetic high-risk status in the adoptee and CD in the adoptive parent ( $OR = 2.00$ , 95% CI [1.14, 3.50]). More importantly, high-risk adoptees who had been reared by adoptive parents with low CD displayed less TD than their low-risk counterparts, suggesting genetic moderation of the environmental risk factor (van Os, Rutten, & Poulton, 2008).

These results were later replicated by the same research group (Wahlberg et al., 2000), who tested 151 adoptees from the same cohort for TD using a different scoring

method (*TDI*, Johnston & Holzman, 1979). Again the relationship between high-risk status and CD was significant ( $OR= 1.70$ , 95% CI [ $1.05, 2.76$ ]). However, at longer follow-up (13 years), Metsänen and colleagues (2007) reported that, whilst the odds ratio for the combination of CD in the adoptive parent and high-risk status in the adoptee was now non-significant in predicting TD ( $OR= 0.21$ , 95% CI [ $0.02, 2.04$ ]), the odds ratio for the low-risk adoptees raised by adoptive parents with high CD was substantial ( $OR= 6.93$ , 95% CI [ $1.30, 36.89$ ]).

Finally, in their most recent publication using largely the same cohort, Roisko, Wahlberg, Hakko, and Tienari (2014) examined the relationships between CD in the adoptive parents and adoptees' TD, schizophrenia-spectrum diagnoses (there were now 12 adoptees with the diagnosis) and season of birth. Interestingly, CD in the adoptive parents was not a predictor of schizophrenia-spectrum diagnosis ( $OR= 0.84$ , 95% CI [ $0.36, 1.95$ ],  $B= -0.17$ , S.E.=  $0.43$ ,  $p= .69$ ) and the same was true for season of birth, genetic risk or any of the interactions between these variables. More importantly, out of all the variables considered (which included adoptees' sex, age, season of birth, genetic risk and the interaction terms) the only significant predictor of TD in the adoptee was parental CD ( $F= 6.99$ ,  $p = .009$ ).

Overall, the evidence suggests an association between CD and TD, which may be specific. However, all studies that have addressed this problem have significant methodological weaknesses, particularly with respect to controlling for co-occurrence of symptoms. Hence, this will be a potentially important avenue for future research. It is worth noting that Wynne and Singer never asserted that CD alone could explain TD in



offspring but rather that it confers a vulnerability which, when combined with later psychosocial stressors, could lead to TD.

In this context, it is important to take note of studies that have reported that TD in patients diagnosed with schizophrenia, and also in patients diagnosed with bipolar disorder, increases significantly when they are asked to talk about stressful topics (de Sousa, Sellwood, Spray, & Bentall, under review; Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995; Tai et al., 2004) and that the same seems to be true for communicational disturbances (Docherty, 1996a). Indeed, the possibility that TD requires some kind of emotional disturbance in addition to CD merits further investigation.

### **6.5.3 Is CD in caregivers associated with relapse in the offspring?**

Only two studies have considered the possible association between parental CD and psychotic relapse. Rund and colleagues (1995) found a significant effect over a 2-year period but, only six families were eligible for inclusion in the analysis, as the experimental task (CCS) required the presence of both parents. In the second study, Velligan et al. (1996) followed-up families of 20 patients diagnosed with schizophrenia for a period of a year following discharge from an inpatient unit. Consistent with Rund et al.'s study, they reported a significant association between parental CD measured at the time of offspring's discharge and offspring's relapse within the following year (relapse was established using BPRS scores and information collected every three months) but not between parental CD measured within the first two weeks of admission and relapse. Velligan and colleagues advanced the hypothesis that CD could lead to relapse by interfering with the family's problem-solving ability, and that cumulative unresolved

problems would increase offspring stress levels, leading to an exacerbation of psychotic symptoms (indeed, they measured CD using transcripts of the family discussing a salient problem). Two other important findings seem to support this hypothesis. First, studies employing problem-solving tasks to measure CD have reported that families of offspring with schizophrenia who have elevated levels of CD are poorer at solving family tasks (Rund, 1986). Second, and as discussed earlier, Lewis and colleagues (1981) reported that parents with elevated CD scores displayed less topic and affective (expression of feelings) focus during family interactions and were more avoidant of eye contact and rigid in their facial expressions. Hence, although CD is usually considered to be a long-term and possibly cumulative influence on the future probability of mental illness, it is conceivable that it may also have an effect on the subsequent course of psychosis once an illness has developed, a possibility that clearly merits further investigation.

#### **6.5.4 Is CD associated with other caregiver characteristics such as expressed emotion or psychopathology?**

Given the association between parental CD and poor mental health outcomes in offspring, researchers have naturally tried to establish whether CD is associated with any other variables in caregivers.

Only a handful of studies have tested if CD is a state- or a trait-like variable. Doane and Mintz (1987) tested 29 young people at two time-points across a time span of 15 years using TAT protocols. The analysis revealed a significant correlation between time-points only for females and only on one factor, *odd or peculiar use of language*.

Velligan and colleagues (1995) tested the mothers of 24 participants diagnosed with schizophrenia on CD whilst they were discussing an emotionally salient problem. The time interval between assessments was 88 days on average. Maternal CD scores across the two time points were moderately correlated, as were offspring's CD scores. However, when the authors corrected the ICD scores for verbosity, the correlation for the mothers dropped to a non-significant level (although a trend was still observed).

In the Finish adoption study, Wahlberg and colleagues (2001) measured CD in 158 participants twice across a time span of 1 year. The participants were divided into a younger (mean age 16) and an older group (mean age 31) and sizable and significant correlations were observed between the two time-points in the latter but not in the former. Contrary to the findings from the Doane and Mintz study, the results were similar for both sexes and across CD categories. In their discussion, Wahlberg et al. suggested that CD might become an enduring and trait-like characteristic in adulthood, stabilizing after adolescence.

The impact of different therapeutic interventions on CD is relevant to this issue of stability over time, and has been examined in three studies. Cole and colleagues (1993) tested the effect of behavioural family therapy and supportive family management on parents of patients diagnosed with schizophrenia. CD ratings were collected at baseline, 16 weeks after therapy and 12 months after discharge. Interestingly, the CD ratings showed a decreasing pattern across time in both treatment conditions (it is worth mentioning that, in this study, CD was not coded but measured using a 4-point Likert scale). Rund and colleagues (1995) used a similar design to test the effectiveness of a two-year psychoeducational program on CD in parents of 12 patients diagnosed with

early-onset schizophrenia. Interestingly, only two parents changed from high- to low-CD across the two time-points, suggesting that this style of communication remained resistant to this psychosocial intervention. In an exploratory trial of the effectiveness of behavioural family therapy on CD against standardised individual treatment, Nugter and colleagues (1997) tested the parents of 52 patients diagnosed with schizophrenia-spectrum disorders across a time-span of 12 months before and after treatment. 65% of the families in the control condition remained high on CD as opposed to 45% in the BFT group. Furthermore, 66% of the mothers in both treatment conditions had remained high on CD across time as opposed to 38% of the fathers. In fact, when both parents were considered, only 17% of the families decreased from high CD at baseline to low CD at follow-up. On the analysis of the different dimensions nearly all CD categories remained stable across time and treatment conditions.

It is possible that, over the short term, CD in the parents of psychotic offspring could be affected by changes in arousal due to test-anxiety or self-consciousness. Schopler and Loflin (1969) reported that parents of psychotic offspring produced less abnormal scores when an object sorting task (OST) was preceded by an interview about their healthy offspring as opposed to when they were interviewed about their psychotic offspring.

Docherty, Sledge, and Wexler (1994) tested parents of psychotic offspring using two 10-minute speech samples. In the control condition, parents were asked to talk freely about pleasant and enjoyable memories and in the experimental condition they were asked to talk about stressful and negative memories. The parents of psychotic offspring, who scored significantly higher on CD than controls, did not produce significantly more

unclear linguistic references in the stressful condition, suggesting that parents' CD was not increased by negative affect. In a subsequent study, Docherty, Hall, and Gordinier (1998) tested 46 parents of patients with schizophrenia and 23 controls using the same methodology. Again, parents of offspring diagnosed with schizophrenia displayed significantly more communication disturbances but their scores, with the exception of *confused references*, did not vary across affective conditions.

Overall, therefore, the available findings suggest that CD is relatively stable in adults, at least over moderate periods of time. On the balance of the limited evidence available, it also appears that CD is not affected by current emotional state. However, it is possible that it is related to more enduring emotional traits, and this has been addressed in studies that have measured the association between CD and expressed emotion.

EE is a variable that represents the amount of negative emotion displayed in the family setting (Brown, et al., 1962; Vaughn & Leff, 1976). The construct has several dimensions but hostility, emotional over-involvement (EOI) and critical comments are thought to be particularly important for offspring psychopathology (Hooley, 2007). In the UCLA high-risk study (Doane, et al., 1981; Goldstein, 1987; Goldstein, 1981, 1985), it was the combination of CD and negative affective style<sup>11</sup> (AS, Doane, et al., 1981) that predicted psychosis in the offspring at both 5 and 15-year follow-up (Goldstein, 1987), with both variables making independent contributions to outcome (Goldstein, 1981; Norton, 1982). Miklowitz and colleagues (1986) later tested the relationship between EE (CFI, Vaughn & Leff, 1976) and CD in 205 parents of offspring diagnosed with schizophrenia-spectrum disorders, reporting that those classified as high-EE had

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<sup>11</sup> Affective style (Doane, et al., 1981) was found to be highly correlated with EE and therefore only the former variable was included in the statistical analysis.

significantly higher CD scores. However, only 16% of the parents in the sample were judged to be both high-CD and high-EE. Nugter and colleagues (1997b) also assessed EE and AS (FMSS, Magaña, et al., 1986; CFI, Vaughn & Leff, 1976) along with CD in parents of psychotic offspring. At baseline and follow-up, no significant differences were found between high- and low-EE parents on CD and CD was not significantly correlated with either EE or AS.

Velligan and colleagues (1990) tested parents of offspring diagnosed with schizophrenia-spectrum disorders with both individual TAT protocols, the ICD along with measures of EE (Magaña, et al., 1986) and AS (Doane, et al., 1981). Neither TAT-CD nor ICD scores were found to be significantly correlated with either AS or EE. In a later study, Docherty (1995a) tested 19 parents of offspring diagnosed with schizophrenia using an adapted version of the ICD (Velligan, et al., 1990), frequency of unclear linguistic references (Rochester & Martin, 1979), TD (TLC, Andreasen, 1986), disorganisation (PANSS, Kay, Fiszbein, & Opler, 1987) along with EE (CFI, Vaughn & Leff, 1976). The only significant differences between high- and low-EE groups were on linguistic reference performance and disorganisation scores and a trend towards a higher prevalence of TD in the high-EE group. Rund and colleagues (1995) found a small but non-significant correlation between the two family variables in parents of 12 offspring diagnosed with schizophrenia-spectrum disorders. More importantly, and as mentioned before, unlike EE (FMSS, Magaña, et al., 1986; CFI, Vaughn & Leff, 1976), CD remained a stable “trait” which was resistant to a 2-year period of family therapy.

In their study designed to test the effectiveness of family therapy on communication and problem-solving skills, Cole and colleagues (1993) tested families of

offspring diagnosed with schizophrenia on CD and EE at baseline and 16-weeks into the program, scoring CD from the verbatim-transcripts of the CFI. Fathers' scores on negative affect (NA) and emotional over-involvement (EOI) were both significantly correlated with scores on CD at baseline. This correlation stood at the 16-weeks' follow-up with CD being again significantly correlated with NA, EOI and critical comments (CC). The mothers' results were quite different with the correlations between CD and EE failing to achieve significance at both baseline and at the 16-weeks follow-up.

Kymalainen (2005; Kymalainen, et al., 2006) tested a multi-ethnic sample of relatives of patients diagnosed with schizophrenia, using the five-minute speech sample (FMSS, Magaña, et al., 1986) to assess CD and the CFI (Vaughn & Leff, 1976) to assess EE, finding a small but, nevertheless, significant association between CD and EE.

However, in a more recent study, Carlson (2011) tested families of psychotic offspring using Kymalainen's CD measure and EE (FMSS, Magaña, et al., 1986) and, in line with Velligan's study, did not find an association between the two variables. Overall, therefore, the association between CD and EE appears to be, at best, weak. A reasonable interpretation of the findings is that CD is a relatively stable characteristic in the speech of parents whereas EE is more sensitive to proximal and contextual variables and can change over time (Santos, et al., 2001; Scazufca & Kuipers, 1998).

It has been argued that CD in parents of psychotic participants might be a sign of psychopathology (Miklowitz & Stackman, 1992), although few studies have addressed this. Of course, the answer to this question will depend on how psychopathology is defined, especially given substantial evidence that psychotic phenomena exist on continua with healthy functioning (Van Os, Linscott, Myin-Germeys, Delespaul, &

Krabbendam, 2009). In this review, we mean by psychopathology simply that the parent meets the criteria for diagnosable mental illness according to conventional criteria such as the DSM (APA, 1987) or the ICD (WHO, 2004).

Wynne and colleagues (1977) reported that CD and the severity of psychopathology in the parents, assessed by experienced diagnosticians who achieved a high degree of inter-rater reliability, were independent and significant predictors of offspring's psychopathology but that CD made a greater contribution. Wender and colleagues (1977) reported no relationship between CD and diagnosable psychopathology (Current and Past Psychopathology Scales, Endicott & Spitzer, 1972) in either biological or adoptive parents.

Drawing on data from the University of Rochester Child and Family Study (URCAFS; Wynne, Cole, & Perkins, 1987), Doane and colleagues (1982) reported that, from all the parents diagnosed with schizophrenia-spectrum disorders (63 out of 145, DSM-III, APA, 1980), only one scored highly on CD. In fact, CD seemed to be more prevalent in parents diagnosed with non-psychotic disorders. Results from the analysis of data from the UCLA high-risk study (Goldstein, 1987) also revealed no relationship between CD and history of psychopathology in the parent (DSM-III, APA, 1980). However, the combination of positive family history of mental health disorder and high CD was present in 86% of the families of offspring with broad-spectrum diagnosis of schizophrenia and in 71% of those with offspring meeting the criteria for a narrow spectrum diagnosis.

Velligan and colleagues (1988) reported no significant differences in CD between mothers who scored high or low on overall psychopathology or on the schizophrenia



subscales of the Minnesota Multiphasic Inventory (MMPI, McKinley & Hathaway, 1943). Hamilton and colleagues (Hamilton, Hammen, Minasian, & Jones, 1993; Hamilton, Jones, & Hammen, 1993) tested 64 mothers of school-aged children; the mothers' diagnoses ranged from depression, bipolar disorder, chronic-ill health to no diagnosis (SADS-L, Endicott & Spitzer, 1978). Again, no significant differences in level of CD were found between the groups. Goldstein and colleagues (Goldstein et al., 1992) tested 56 parents of psychotic offspring and again found no relationship between high levels of CD and presence of diagnosable mental health disorder in the parents (DSM-III-R, APA, 1987).

Docherty (1993) reported a significant correlation between CD and schizotypy in parents assessed using the Schedule for Schizotypal Personalities (SSP, Baron, Asnis, & Gruen, 1981). In a later study with colleagues (1998) she reported a significant association between communication disturbances in parents of offspring diagnosed with schizophrenia, and family history of psychosis and schizotypy in first degree relatives of the parents. These findings were later replicated by the same research group (Docherty et al., 1999) who used a communicational index that targets disturbances at level discourse deixis. It is worth mentioning that schizotypy, although a personality dimension that has been linked to schizophrenia (Lenzenweger, 2006), is not itself regarded as a diagnosable form of mental illness. Finally, Subotnik and colleagues (2002) tested the relationship between family history of mental health problems and CD in 89 parents. They reported that mothers with family history of schizophrenia had significantly higher CD scores than mothers without such history but no significant differences were found for family history

of affective disorders. No significant effects were found for the group of fathers with either the family history of schizophrenia or affective disorders.

Finally, four studies have addressed whether CD might be a culture-bound construct by carrying out assessments across different ethnicities and cultures. Behrens, Rosenthal, and Chodoff (1968) found no significant differences in CD between 56 African- and White-American parents of offspring diagnosed with schizophrenia. Similar results were obtained when Doane and colleagues (1989) compared 64 carefully matched Mexican- and Anglo-American families of offspring diagnosed with schizophrenia using individual TAT protocols. In a more recent study, Kymalainen et al. (2006) tested a sample of 57 White, Latino and African-American relatives of participants diagnosed with schizophrenia. CD was tested using the five-minute speech sample (FMSS) and a well established CD scoring system (Velligan, 1985). White and Black relatives showed more CD than did Latinos, but no other significant differences were observed. In contrasting this study with previous findings, the aspect that becomes more salient is that Kymalainen and colleagues sample had included relatives other than parents.

In an unpublished dissertation, using the same methodology, Carlson (2011) tried to adjudicate between these conflicting results by assessing 85 relatives of White, Black and Hispanic-Americans diagnosed with schizophrenia and reported no significant differences between the ethnic groups, replicating the earlier findings from Doane and colleagues.

Overall, the findings from studies of the association between CD and other caregiver characteristics are remarkable for what was not observed. CD seems to be a cross-culturally valid, relatively stable trait that does not change over time. It is not

affected much by current emotional state, nor related to expressed emotion, a measure of emotional functioning within the family that can change over time. Nor is CD directly related to psychopathology in the parent, although it does seem to be associated with a family history of psychosis and also with schizotypal traits in parents.

### **6.5.5 Can the association between CD in the caregiver and offspring psychopathology be explained by reverse-causality (the caregivers' reactions to their disturbed children)?**

Some authors have suggested that CD in the parent could be a response to the offspring's psychosis or other characteristics (Liem, 1974; Miklowitz & Stackman, 1992; Mishler & Waxler, 1968) and this issue has been considered by researchers in a number of ways.

One approach has been to address the issue of temporal precedence over the long term. The only investigation that has attempted to investigate this was the UCLA high-risk study (Goldstein, 1987) given that none of the 64 young people who were recruited were psychotic at baseline. As mentioned before, at both 5-year (Doane et al., 1981) and 15-year follow-up (Goldstein, 1987), parental CD alone and in combination with parental negative AS were significant predictors of broad-spectrum schizophrenia diagnoses in the offspring (DSM-III-R, APA, 1987). Unfortunately, this study stands as the only prospective study of its kind.

A second approach has been to examine whether CD is found only when parents are interacting with their psychotic offspring. Feinsilver (1970) tested parents of offspring diagnosed with schizophrenia and healthy controls using a task where parents and siblings had to describe an object without naming it. Consistent with previous research,

Feinsilver reported that parents of psychotic offspring evidenced significantly more CD than the comparisons. More importantly, these parents displayed CD whether they were talking to their psychotic or to healthy offspring, suggesting that CD is not contingent on psychopathological characteristics of the offspring.

Some studies have examined interactions between parents and psychotic offspring in detail. Holte and Wichstrøm (1990b) used the colour conflict method (CCM see Table 16), comparing families of offspring diagnosed with schizophrenia, with families with other mental health diagnoses and healthy offspring. This task is especially relevant as it allows the experimental manipulation of the degree of perceptual conflict (difference of the colour saturation between the coloured plates) during the interactions. Amongst the three groups, offspring diagnosed with schizophrenia displayed a significant increase in *egocentric utterances* towards their parents across conditions. More importantly, the parents of offspring diagnosed with schizophrenia produced significantly more *active disqualifying communications* (throwing doubt upon the other persons' status, credibility or confidence) towards their offspring and partners in the conflict condition compared with the comparisons. Unfortunately, this study did not provide a sequential analysis of the communication and therefore did not address the issue of the direction of causality (it is plausible that the increase in *egocentric utterances* in the offspring could have driven the increase of *active disqualifying communication* in the parent or vice versa).

In a second study using sequential analysis, Wichstrøm and Holte (1992) analysed a large number of utterances of parents during the Consensus-Spouse Rorschach (CSR, Loveland et al., 1963) using the same three parental groups. The researchers reported that, amongst parents of offspring diagnosed with schizophrenia, the production of a *self-*

*disqualification* (utterances that are vague, egocentric or paradoxical) in one of the spouses increased the probability of the occurrence of a *self-disqualification* in the partner by nearly fifteen times ( $OR= 14.55, p< .0001$ ). This is especially relevant given that *self-disqualification* and *disqualifying communication* in parental communication are significant predictors of anxiety (Wichstrøm, Holte, & Wynne, 1993), social competence (Wichstrøm, Anderson, Holte, & Wynne, 1996; Wichstrøm, Holte, Husbey, & Wynne, 1994) and poor social-emotional functioning in at risk young children (Wichstrøm, Anderson, Holte, Husby, & Wynne, 1996). Unfortunately, the study did not include the offspring's utterances in the sequential analysis; we are aware of no studies that have attempted this to date.

A final approach has been to investigate whether CD is related to specific characteristics of the child or the parent. Roisko and colleagues (2011), using data from the already mentioned Finnish high-risk adoption study, measured CD in the adoptive parents using both individual Rorschach protocols and the family Rorschach (where offspring is present), and TD in both high- and low-genetic risk adoptees using the thought disorder index (TDI, Johnston & Holzman, 1979). CD in the individual Rorschach protocols of adoptive mothers and fathers was unrelated to the adoptees' age, sex, risk status, CD (individual Rorschach) or TD scores. In the family Rorschach, maternal CD scores were associated with the adoptees' age ( $B= -0.41, p= .02$ ) and TD ( $B= 0.31, p= .016$ ) and CD scores in the individual protocols ( $B= 0.54, p< .0001$ ) but also, and not surprisingly, with the adoptive mother's own score in the individual Rorschach ( $B= 2.54, p< .0001$ ). The adoptive fathers' scores in the family Rorschach were associated with adoptees' age ( $B= 0.48, p= .007$ ), age of the adoptive father ( $B= -$

0.29,  $p = .045$ ) and again, unsurprisingly, the fathers' own scores on the individual Rorschach ( $B = 3.11$ ,  $p < .0001$ ). In summary, these results suggest that parental CD is more strongly associated with characteristics of the parents than with characteristics of the adoptee. Regarding the associations between adoptive mothers and adoptees CD in the family Rorschach, Roisko et al. suggest that:

“It is also possible, and even probable, that maternal CD has in fact a greater effect on the children because mothers spend more time with their children than fathers, or at least they did at the time when the adoptive children in this series were growing up (...) This finding fits with the assumption that mothers have a more intensive relationship with their children than fathers, so that a reciprocal interaction between the CD of the adoptive mother and the child is thus more probable than between the father and the child.” (pp. 66-67).

Overall, the results of these studies seem to suggest that CD is an independent parental characteristic that precedes the onset of psychosis and is not contingent on specific characteristics of the offspring. However, it is likely that, at a certain point along the developmental pathway to the offspring's mental illness, parent and offspring's communication difficulties become intertwined, each affecting the other as seems to be true in the case of EE (Cook, Strachan, Goldstein, & Miklowitz, 1989). However, this hypothesis needs to be examined in future studies.

### **6.5.6 Can the association between CD in the caregiver and offspring psychopathology be explained by genetic factors?**

Some authors have interpreted the findings of the research on CD as evidence of shared genetic vulnerability in the family (Kinney et al., 1997; Miklowitz & Stackman, 1992; Wender et al., 1977) and some have even suggested a specific genetic *loci* for that vulnerability in chromosome 7 (e.g. specific variants of the *FOXP2-CNTNAP2* genetic pathway, Levy et al., 2010) for both CD and TD. A genetic association between CD and psychosis is certainly plausible because, as we have seen (see Section 4) CD in parents, although not associated with parental mental illness, is associated with schizotypal traits in parents and with a family history of mental illness. However, other evidence suggests that genetic confounding is unlikely to explain the richness and robustness of the association between parental CD and offspring psychopathology, and that any genetic contribution is likely to be complex.

Two early studies addressing this question gave inconsistent findings. Wynne and colleagues (1976) reported no statistical differences between the biological and adoptive parents of offspring diagnosed with schizophrenia, with both groups of parents obtaining significantly elevated CD scores compared to a control group of adoptive parents of healthy offspring. However, Wender and colleagues (1977) also compared adoptive and biological parents of psychotic offspring using Rorschach protocols and reported that the biological mothers scored significantly higher than the adoptive mothers. Unfortunately, this latter study was compromised by a poor inter-rater reliability of the CD ratings.

The research program that has most informed this debate is the Finnish adoptive family study (Tienari et al., 1985) which has been mentioned several times earlier in this

review, and which recruited the offspring of biological mothers diagnosed with schizophrenia-spectrum disorders (DSM-III-R, APA, 1987) and the offspring of biological mothers with no such mental health history. Adoptees were on average 15-months old when they were placed with the adoptive family (and they were all adopted before reaching the age of 4), so that exposure to the biological family was limited.

As discussed earlier, Wahlberg and colleagues (1997) found that TD increased steeply in genetically high-risk adoptees as CD increased in the adoptive parent and that high-risk adoptees who had been reared by adoptive parents with low CD displayed less TD than the low-risk adoptees, supporting a “corrective parent” model (i.e. that functional communication in the parents may counteract the inherited vulnerability of the offspring, Doane, 1978). These results were later replicated by the same research group (Wahlberg et al., 2000) using a different method to score TD (TDI, Johnston & Holzman, 1979).

As discussed earlier, at 13 years follow-up, Metsänen and colleagues (2007) reported that, whilst the association between adoptive parent CD and offspring TD was now non-significant for the high-risk adoptees, the association for the low-risk adoptees had become substantial. Hence, at this follow-up, it seemed that adoptive parental CD was associated with an increased risk of TD in the genetically non-vulnerable offspring. This finding seems surprising in the light of the earlier results from the same study, but does not detract from the overall evidence that exposure to CD in either a biological or non-biological parents increases the risk of TD suggesting an environmental effect.



## **6.6 Conclusions from the review**

### **6.6.1 Limitations of CD research to date**

We have reviewed a sizeable volume of research on CD that has accumulated since the 1960s. Before reaching an overall conclusion, it is important to note some limitations in the existing research. The first is that many of the studies were undertaken long before the publication of DSM-III and used diagnostic approaches that were quite different to today's standardised methods.

Also, the majority of the case-control studies have been limited to two groups and have not considered other relevant mental health diagnoses in their designs. For example, we have found only one study that directly compared parents of patients diagnosed with bipolar disorder and the parents of offspring diagnosed with schizophrenia. Furthermore, there is considerable variability in how CD was measured and quantified (*e.g.* some studies calculate a CD ratio using lines of speech whereas others used word count) and, in some of the earlier studies, inter-rater reliability data were not published.

Regarding the potential association between CD and TD (as opposed to psychiatric diagnosis), the studies have been sparse and have not adequately controlled for symptom co-morbidity. This is an important limitation given that CD and other patterns of communication in the parent have been reported to be associated with other outcomes in the offspring such as social maladjustment (Albers, Doane, & Mintz, 1986; Doane et al., 1982; Siira, Wahlberg, Hakko, Läksy, & Tienari, 2007; Siira, Wahlberg, Hakko, & Tienari, 2013).

In future studies, it will be important to consider not only specific associations but also more complex pathways between parental characteristics and outcomes in their

children. For example, to date, no work has been carried out to examine associations between CD and social predictors of psychosis (e.g. relative deprivation), psychological mechanisms (e.g. social cognition), or neural substrates (e.g. areas associated with TD such as the dorsolateral prefrontal cortex, Cohen & Servan-Schreiber, 1992; Perlstein, Carter, Noll, & Cohen, 2001, or areas associated with social cognition such as the medial prefrontal cortex and bilateral posterior temporo-parietal junction, Schurz, Radua, Aichhorn, Richlan, & Perner, 2014).

### **6.6.2 The possible causal role of CD**

Nevertheless, despite these limitations, it is possible to reach a general conclusion about the likely causal role of CD in offspring's psychosis. In this context, it is important to recognize that there are substantial philosophical debates about the inference of causality (Mumford & Anjum, 2013) and that different criteria have been proposed for making this inference. Within the epidemiological literature, Hill's (1965) criteria are most commonly cited. These are: (a) *strength* of association; (b) *consistency* of the findings; (c) *specificity* of the effects; (d) *temporal* precedence; (e) *dose-response* relationship; (f) *plausible* explanation (or mechanism); (g) *coherence* of the finding with present knowledge; (h) "occasionally it is possible to appeal to experimental or semi-experimental evidence"; and, (i) similar findings in *analogous* studies (e.g. animal studies). These criteria are not necessary or sufficient conditions to infer causality but rather a flexible framework to test confidence in the interpretation of the direction of the statistical effect.

The studies reviewed in the previous sections suggest that the association between CD in the caregiver and psychosis in the offspring is both strong and consistent satisfying

the first two criteria. Moreover, our recent meta-analysis yielded a hedges'  $g$  of large magnitude ( $\geq .8$ , Cohen, 1988) with the studies reviewed showing that different research groups using different methodologies have *consistently* replicated Wynne and Singer's original findings. Evidence from a more limited set of studies have indicated that CD takes temporal precedence over the onset of psychosis in offspring and that effect-sizes are not attributable to genetic confounding (or other confounders). Moreover, some studies have reported evidence of a *dose-response* relationship between parental CD and severity of psychosis in the offspring; however there is also some evidence that CD is, at least to a degree, *specifically* associated with TD. This latter finding could potentially explain why elevated CD scores have also been found in parents of offspring diagnosed with bipolar disorder, although no study to date has adequately controlled for symptom co-morbidity. The potential causal relationship between CD in the caregiver and TD in the offspring is *coherent* with increasing evidence that the environment plays an important role in psychosis (Bentall & Fernyhough, 2008; van Os, Kenis, & Rutten, 2010).

Some of Hill's criteria (for example, evidence from experiments and analogous studies) are likely to be difficult to meet in the case of CD (it is not obvious how long-term exposure to CD might be experimentally manipulated, for example). Nonetheless, confidence that CD has a causal impact on the disruption of psychological wellbeing of offspring will undoubtedly be increased by the identification of plausible mechanisms linking the two, which we will now consider.

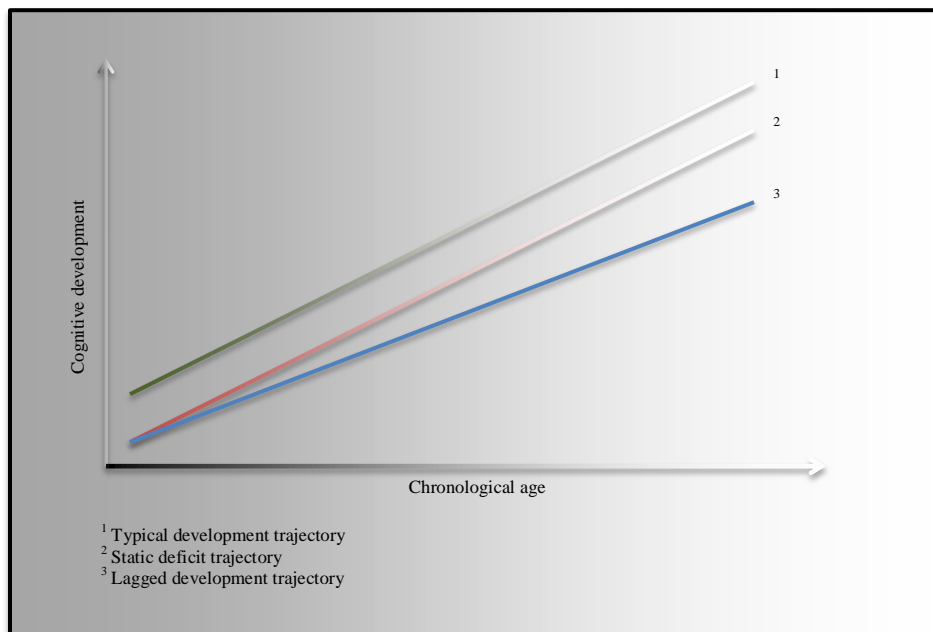
### 6.6.3 Possible developmental pathways from CD to offspring psychopathology

It is a well-replicated finding that the diagnosis of schizophrenia in adulthood is associated with cognitive difficulties (Ellevåg & Goldberg, 2000) and that these difficulties, despite being subtle, are already identifiable in childhood (Welham, Isohanni, Jones, & McGrath, 2009). For example, several birth cohort studies have identified poor performance on cognitive tests and indices of scholastic functioning in the early childhood of individuals who are later diagnosed with schizophrenia-spectrum disorders (Cannon et al., 2002; Cannon et al., 2006; Done, Crow, Johnstone, & Sacker, 1994; Jones, Rodgers, Murray, & Marmot, 1994; Kremen et al., 1998; Niendam et al., 2003; Osler, Lawlor, & Nordentoft, 2007) as well as more specific language and speech difficulties in children as young as two (Bearden et al., 2000; Cannon et al., 2002; Crow, Done, & Sacker, 1995; Jones et al., 1994). In one of these cohort studies unintelligible speech at the age of 7 was found to be significantly associated with later diagnosis of schizophrenia ( $OR= 12.7$ , 95% CI [2.46, 65.66], Bearden et al., 2000).

These cognitive deficits may take different developmental trajectories. For example, whilst some seem to be present since childhood and remain stable throughout development (*developmental deficit model* see Figure 6), others are indicated by a growth in cognitive function that lags behind that of typically developing children (*developmental lag model* see Figure 6, Reichenberg et al., 2010).

The atypicalities reported in birth cohort studies have generally been interpreted as evidence for a neurodevelopmental framework in which the development of schizophrenia is thought to be predisposed by a combination and interaction of polygenetic factors and early pre- and perinatal non-psychological insults to the CNS

(e.g. *placental pathology* or *toxoplasma gondii infection*, Murray & Lewis, 1988; Rapoport, Addington, & Frangou, 2005; Rapoport, Giedd, & Gogtay, 2012). An alternative hypothesis, but by no means incompatible, is that specific atypicalities in the way the caregiver relates and communicates to the child may contribute to the cognitive atypicalities often observed in children who later develop psychotic experiences (Wynne, 1984). Several strands of evidence support the plausibility of this account.



**Figure 6** - Schematic representation of two different developmental models of schizophrenia (Reichenberg et al., 2010).

First, studies with healthy children and their families have shown that developmental and psychological mechanisms that may be important in psychosis (e.g. Theory of Mind skills in TD, Brüne, 2005; Sprong et al., 2007) are open to environmental influence (Sabbagh & Callanan, 1998; Peterson & Siegal, 2000; Hughes et al., 2005). Indeed, several social factors have been associated with healthy differences in social-

cognitive development. Factors associated with good social-cognitive development include having older siblings (Cassidy, Fineberg, Brown, & Perkins, 2005; Ruffman, Perner, Naito, Parkin, & Clements, 1998) and older family members (Lewis, Freeman, Kyriakidou, Maridaki-Kassotaki, & Berridge, 1996); exposure to family communication about feelings and causality (Dunn, Brown, Slomkowski, Tesla, & Youngblade, 1991; Dunn, Brown, & Beardsall, 1991); maternal talk about mental states (Meins et al., 2002); parents' employment of disciplinary strategies that focus on mental states (Ruffman et al., 1999); and the caregiver's capacity to reflect about the child's internal experience (Oppenheim & Koren-Karie, 2002).

Second, CD in mothers of psychotic offspring has been found to be significantly associated with the offspring's poor performance on neuropsychological tasks (e.g. span of apprehension test or the continuous performance test) that measure sustained attention and distractibility (Asarnow et al., 1988; Nuechterlein, Goldstein, Ventura, Dawson, & Doane, 1989; Rund, 1985; Wagener, Hogarty, Goldstein, Asarnow, & Browne, 1986); the same difficulties have been found to be associated with TD (Kerns & Berenbaum, 2002) and communication disturbances in patients diagnosed with schizophrenia (Docherty, 2005). High-risk longitudinal studies have also reported significant associations between CD in caregivers and poorer cognitive development and problem-solving ability (Fisher & Jones, 1980) and poorer performance on IQ tests in the child (Greenwald, 1989).

One area of environmental influence that has been very rarely explored (at least appropriately) in the field of psychosis is the early relationship between caregiver and child. This is surprising given that there is now accumulating evidence that attachment

plays an important role in a range of outcomes in psychotic patients (for a review see Gumley, Taylor, Schwannauer, & MacBeth, 2014). Consistent with this, in one birth cohort study, health visitor's rating of the mother as having below average maternal skills and understanding of her child at age of four increased the likelihood of a diagnosis of schizophrenia in adulthood ( $OR= 5.8$ , 95% CI [0.8, 31.8], Jones et al., 1994) and in another, the mothers of children later diagnosed with schizophrenia-spectrum disorders were significantly more likely to have atypical mother-child interactions ( $OR= 2.65$ , 95% CI [1.2, 5.6], Cannon et al., 2002). Furthermore, in a more recent longitudinal study, maternal EE was found to be a significant predictor of psychotic experiences in the child ( $\beta= 0.24$ ,  $p= .02$ , Polanczyk et al., 2010).

The findings from these studies are consistent with the findings from studies showing specific social-cognitive difficulties in the healthy relatives of patients. A recent meta-analysis examined a range of different social-cognitive domains in parents, sibling and offspring of patients diagnosed with schizophrenia, reported significant and consistent effect-sizes for specific difficulties in *mentalising* ( $d= 0.48$ ), *emotional processing* ( $d= 0.41$ ), and *social perception* ( $d= 0.42$ ) (Lavoie et al., 2013). A sub-analysis using just data from parents revealed an effect-size of the same magnitude ( $d= 0.65$ ). Moreover, in a recent case-control study carried out by the same group, Lavoie and colleagues (2014) reported that the parents of psychotic offspring performed significantly worse on mentalisation ( $d= -0.64$ ) than controls and this difference remained significant after controlling for IQ. Other studies have also reported that parents of psychotic offspring have difficulties recognizing affect from facial expressions (McCown, Johnson, Austin, & Shefsky, 1989) and perform worse on ToM tasks (Anselmetti et al., 2009).

Consistent with this, and as already noted, we also reported in our meta-analysis that CD was significantly more prevalent amongst mothers of offspring diagnosed with schizophrenia-spectrum disorders (de Sousa et al., 2013). Therefore, it is conceivable that CD and mentalising difficulties in the caregiver could have an impact on child's early development.

Several authors have suggested that if this was the case then the impact of CD would be likely to occur in the early stages of child development (Rund, 1985; Velligan et al., 1988; Wynne, 1984). Such an effect might be explained by the disruption of a number of developmental mechanisms.

#### 6.6.3.1 Joint attention (JA)

First, given that CD is defined as a difficulty in the establishment and maintenance of a focus of attention during communication (Wynne, 1981, 1984) and that it is associated with distractibility in parents of patients diagnosed with schizophrenia ( $r = 0.51$ ,  $p = .03$  Docherty, 1993) one possible developmental mechanism of action is through its potential impact on early episodes of joint attention (JA).

JA refers to relational and referential episodes of shared attention by which the child *engages* with the caregiver in a sequence of timely and synchronous communicative exchanges focused on an external referent (*e.g.* object, event or activity) through eye gazing, pointing and gestures or through verbal and other non-verbal means (Mundy & Jarrold, 2010; Scaife & Bruner, 1975; Mundy & Newell, 2007; Tomasello et al., 2005).

The child's ability to engage in full JA episodes is believed to be established by 15-24 months of age (Bakeman & Adamson, 1984; Charman et al., 2000) building up



from the early protodialogues between caregiver and child (Tomasello et al., 2005) and possibly driven by reward-related brain areas (*e.g. ventral striatum*, Schilbach et al., 2010).

More than just converging lines of sight, a set of skills (Butterworth & Jarrett, 1991) or an in-built module in the brain (Baron-Cohen, 1995), JA refers to an episode of intersubjectivity and mutual *self-other-consciousness* that anchors the child's development (Trevarthen & Aitken, 2001). Numerous studies have documented the role of JA in fostering more complex social and cognitive abilities (Mundy & Newell, 2007) such as executive function (Mundy & Newell, 2007; van Hecke et al., 2012), ToM (Charman et al., 2000; Nelson, Adamson, & Bakeman, 2008), social and emotional functioning (Nowakowski, Tasker, & Schmidt, 2012; Van Hecke et al., 2007), emotion regulation (Morales, Mundy, Crowson, Neal, & Delgado, 2005), language acquisition (Markus, Mundy, Morales, Delgado, & Yale, 2000; Morales et al., 2000; Morales, Mundy, & Rojas, 1998; Mundy et al., 2007; Tomasello & Farrar, 1986) and conversational skill (Farrant, Maybery, & Fletcher, 2011).

The important developmental aspect of JA seems to be its *sustainability* through timely, shared intentional communicative exchanges between the dyad introducing basic principles of “phrasing”, “narrative”, turn taking and therefore promoting cognitive development (Adamson et al., 2014; Tasker & Schmidt, 2008).

These episodes between child and caregiver around an external referent are believed to facilitate depth of information processing (Striano, Reid, & Hoehl, 2006) indexed by neural correlates of attentional processing in event-related potential studies (Hirotani, Stets, Striano, & Friederici, 2009; Striano et al., 2006) and to facilitate parallel

processing between distal distributed and integrated neural networks of frontal temporal and parietal cortical and subcortical neural activity (Mundy & Jarrold, 2010).

Functional magnetic resonance imaging studies contrasting joint versus non-joint attention, have reported associations between JA and activity in the ventromedial frontal cortex, the left superior frontal gyrus, cingulate cortex, and caudate nuclei (Williams, Waiter, Perra, Perrett, & Whiten, 2005) and the dorsal medial prefrontal cortex and right posterior superior temporal sulcus (Redcay, Kleiner, & Saxe, 2012). Studies using near-infrared spectroscopy have reported that the left dorsal prefrontal region seems to be specifically sensitive to JA interactions (Grossmann & Johnson, 2010) and that there are significant differences in functional connectivity of the frontal areas in response to JA versus non-JA stimuli (Zhu, Yadav, Rey, & Godavarty, 2009).

The interesting aspect of these studies is that they support the hypothesis that the brain-areas that are recruited for JA are the same areas that are involved in ToM skills and mentalization. More importantly, the progressive coordination of self-other attention is believed to become internalized, through shared practice of routines (Racine & Carpendale, 2007), into a self-organising system that allows human beings to maintain covert attention to common abstract representations enabling therefore, symbolic development and thought (Mundy & Jarrold, 2010; Mundy & Newell, 2007; Tomasello et al., 2005).

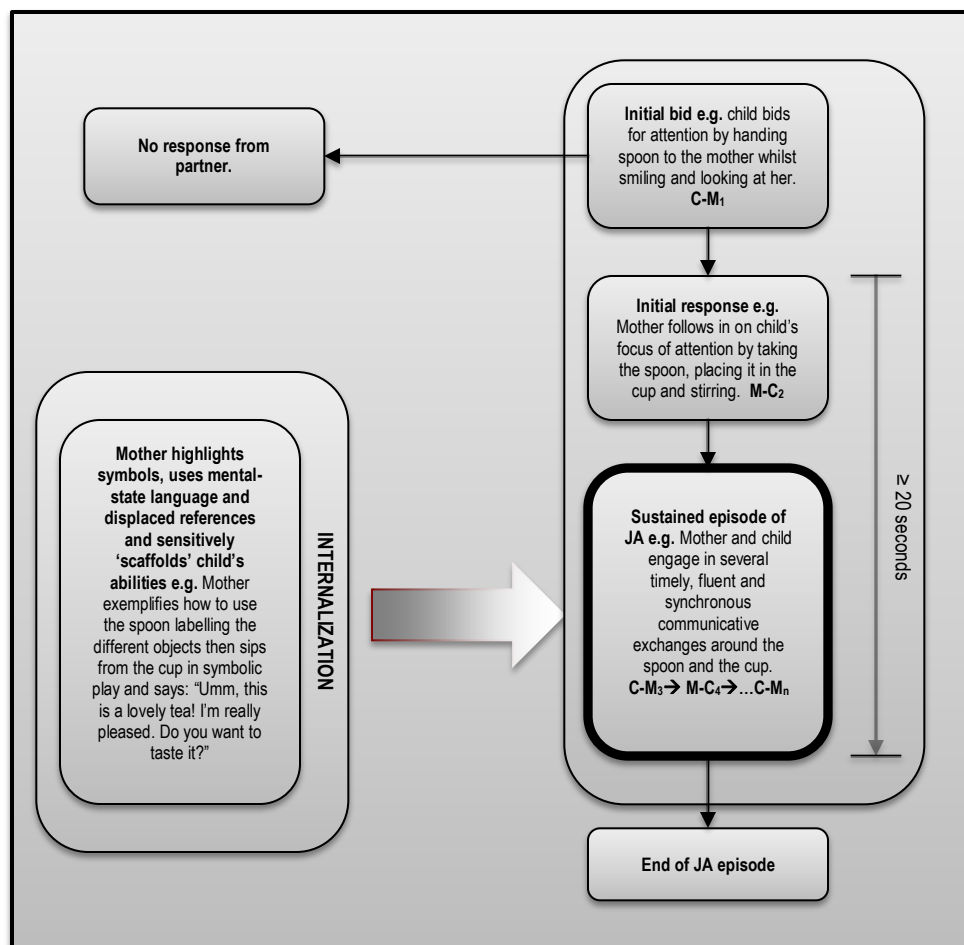
These episodes of JA are of course influenced the mother's interactive style (Roberts et al., 2013; Tasker & Schmidt, 2008; Tomasello & Todd, 1983; Vaughan et al., 2003). For example, factors such as depression in the mother have been shown to be negatively associated with mother's engagement in bouts of coordinated joint

engagement with the child (Goldsmith & Rogoff, 1997a). Consistent with this, one study reported that maternal sensitivity was a significant predictor of the child's propensity to engage in episodes of joint attention (Hobson, Patrick, Crandell, Perez, & Lee, 2004) and other studies have documented the importance of the caregiver's ability to follow in on the child's focus of attention (Bono & Stifter, 2003; Carpenter, Nagell, & Tomasello, 1998; Mendive, Bornstein, & Sebastián, 2013; Tomasello & Farrar, 1986).

Tasker and Schmidt (2008) have proposed an interesting reconceptualization of the JA construct in which the entire focus is on the exchanges that occur *within* the episode in what the authors called *consummative* JA. Figure 7 shows an example of JA where we highlight this important feature. The emphasis of the proposal is on the *span* and *quality* of the communicative exchanges *within* a sustained episode. In the authors' conceptualisation, an episode that is not sustained through synchronous and timely exchanges after its establishment would be considered an *empty* episode of JA. One possible hypothesis is that CD may impact on the child's cognitive development by disrupting these early episodes of JA.

#### 6.6.3.2 Internalization of inner speech

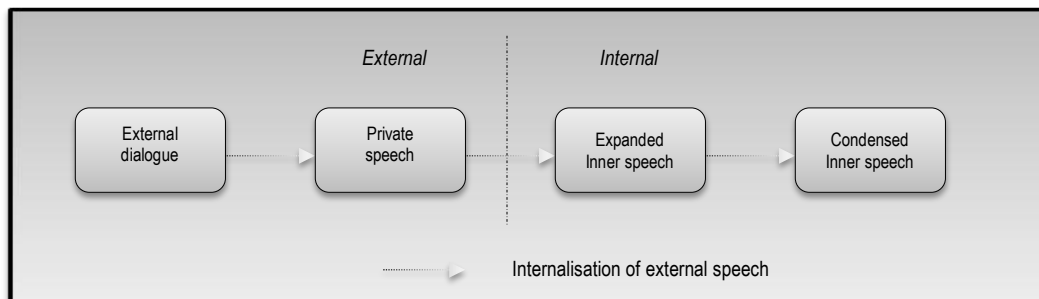
Another possible developmental mechanism that could explain the environmental effect of CD on the offspring's cognition is through the process of *internalization* of external dialogue that is vague, ambiguous or contradictory and the consequent disruption of cognitive development as proposed by Rund (1985).



**Figure 7 - Example of joint attention.**

According to Vygotsky, the cognitive development of the child occurs through a process of internalization of external speech (that progressively becomes covert and condensed through a process of syntactic and semantic transformations - see Figure 8). Vygotsky suggested that the development of the voluntary control of the attentional processes in the child takes place in early infancy through the child's *internalization* of the consistent action of caregivers who lead and direct the child's attention to the surrounding stimuli through verbal and non-verbal means.

Using a Vygotskian framework, Fernyhough (2004) suggested that, in psychosis, especially when the patient experiences hallucinations, there may be disruptions in the process of internalization of external speech due to an impoverished two-way dialogue with the caregiver. Such disruptions could be explained by disruptions at the level of attachment in the dyad. However, another possibility is that CD and poor deictic referencing in the speech of the caregiver may disrupt these early dialogues. For example, a different body of research has reported that a rich and elaborative maternal style of reminiscing is associated with a range of positive socio-cognitive and emotional outcomes in the child ranging from strategic memory, language and literacy development (Fivush et al., 2006).



**Figure 8** - Vygotsky's (1934) model of the development of verbal thought and higher cognition.

#### 6.6.3.3 Mind-mindedness

Not incompatible with these hypotheses is the idea that CD could potential impact on the offspring's development via *maternal mind-mindedness*. Mind-mindedness refers to the caregiver's ability to use of language that appropriately reflects the offspring's mental state. There is now a growing body of work supporting the role of maternal mind-mindedness as an early predictor of ToM development in the offspring (Laranjo, Bernier,

Meins, & Carlson, 2010; Meins et al., 2002; Meins & Fernyhough, 1999; Meins et al., 2003) as well as joint attention (Roberts et al., 2013) and attachment security (Meins, Fernyhough, Fradley, & Tuckey, 2001). Although a relationship between CD and maternal mind-mindedness is still to be tested it is conceivable that a fragmented, contradictory or vague style of communication could interfere with the caregiver's ability to appropriately label the offspring's mental states and consequentially have a negative impact on the offspring's social-cognitive development.

Furthermore, over recent years there has been a renewed interest amongst researchers on the social origins of executive function in young children (Carlson, 2009). It is becoming clear that parenting behaviours such as maternal mind-mindedness and maternal sensitivity amongst others seem to play a crucial role in the early development of the child's executive function (Bernier, Carlson, & Whipple, 2010). Some work has also been developed examining the relationship between parental scaffolding (infant-centred, sensitive and supportive behaviours that facilitate child's achievement of a goal that would likely not occur without the support) during the child's goal-directed activity and early development of executive function (Hughes & Ensor, 2009). For example, timing and contingency of parental utterances that are conceptually rich and not directive seem to have a significant impact on child's attention-switching executive ability (Bibok, Carpendale, & Müller, 2009). This is hardly surprising given that components of executive function start to develop at a very early age (Best & Miller, 2010) and the prefrontal cortex is amongst the slowest areas to develop in terms of synaptogenesis and myelination (Romine & Reynolds, 2005) providing a wider window for the impact of socio-contextual factors.

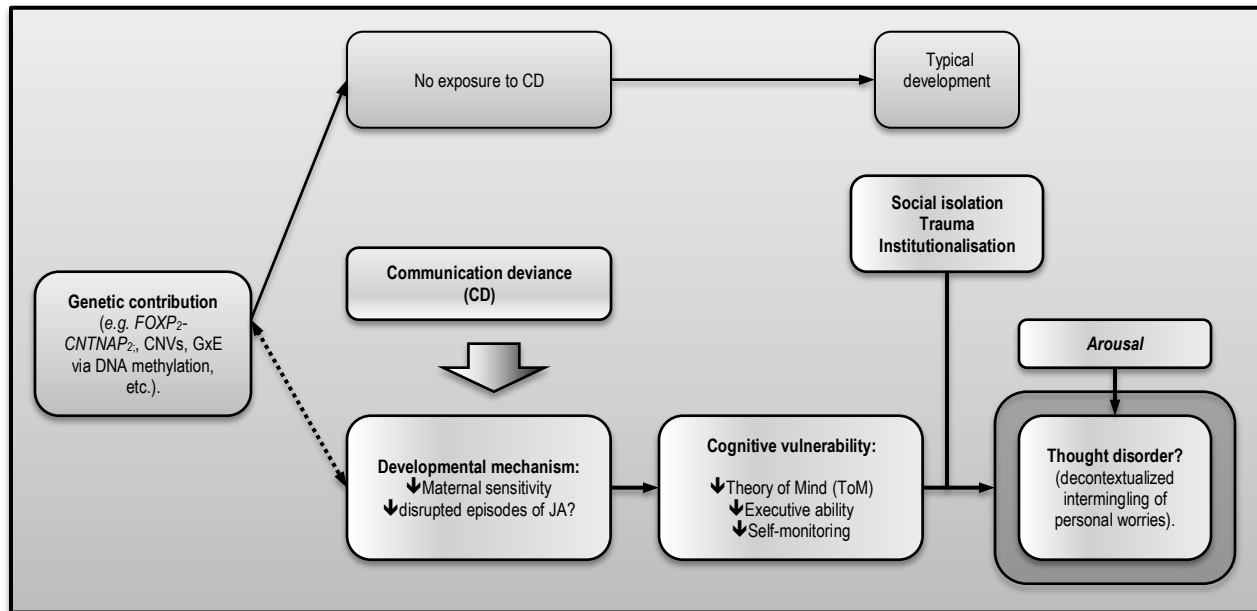
Our proposal is that CD could potentially impact on the caregiver's ability to provide timely and contingent elaborative utterances and therefore impact on the development of executive ability, which has been shown to be a cognitive domain of importance to schizophrenia (Fioravanti, Carlone, Vitale, Cinti, & Clare, 2005) and which is especially associated with TD (Kerns & Berenbaum, 2002; Stirling, Hellewell, Blakey, & Deakin, 2006).

#### **6.6.4 Conclusions about possible developmental pathways**

The biggest challenge ahead is to explore developmental pathways from CD to psychosis and TD. We have presented several plausible and testable mechanisms and developmental pathways to explain how the caregiver's CD may impact on the child's early cognitive development. In Figure 9, we present a tentative and hypothetical developmental pathway that accounts for some predictions. We propose that if CD in the caregiver affects the child's early socio-cognitive development via JA than we should be able to observe an association between CD and a lower sustainability and shorter span of communicative exchanges within the JA episodes leading to fewer opportunities for the caregiver to highlight symbols and scaffold the child's early development. Another concurrent prediction is that high CD caregivers may have difficulties "following in" on the child's focus of attention given that CD reflects basic difficulties sharing and *maintain* a focus of attention. Such difficulty could potentially explain difficulties responding and sustaining the child's bids for attention.

Not precluded is the hypothesis that high CD may be a more cross-modal expression on the caregiver's inability to communicate in a broad sense and respond

appropriately and sensitively to the pre-verbal child's needs. If such a relationship exists, we should observe an association between CD and maternal insensitivity to child's distress and non-distress as well lower fluency and connectedness in the dyad at an earlier stage. Exploring this relationship is important given the large body of literature suggesting that disrupted maternal communication in early development is associated not just with less time spent in JA (Hobson et al., 2004) but also with dissociative experiences later in life (Dutra et al., 2009) and a range of other psychopathological outcomes in the offspring (Lyons-Ruth, 2008).



**Figure 9** - A very hypothetical developmental pathway between CD and thought disorder.

It would be pertinent to test these relationships using a cohort of primiparous mothers at socio-economic risk following them up from pregnancy to children's early childhood. CD could be measured before the birth of the first child in the mothers using five-minute speech samples so that behavioural correlates of CD during mother-infant



interaction could be explored independently. One exploratory hypothesis is that mothers with high CD scores measured during pregnancy will show less sensitivity to their infants' displays of distress and needs. Also if our predictions are correct, these dyads will display less engagement during bouts of JA and caregivers will show fewer relevant behaviours such as scaffolding and symbol highlighting during interaction with their children at a later stage.

Obviously, it would be very time-consuming and expensive to follow up these offspring from childhood to adulthood to then screen them for psychotic experiences. Also, schizophrenia has a small prevalence in the population (4.0 per 1,000, McGrath, Saha, Chant, & Welham, 2008) and although subclinical psychotic experiences are more prevalent in the general population (Hanssen, Bak, Bijl, Vollebergh, & van Os, 2005) the likelihood is that in a medium size study these populations would be grossly underrepresented.

One way to circumvent this obstacle would be use retrospective data of the early development (e.g. video recordings) of adults with psychotic experiences. For example, Walker and Lewine (1990) used home videos to study the early childhood of five patients diagnosed with schizophrenia. Unfortunately, such strategy is limited by the fact that the observed and rated situations were not standardised (and by the low numbers of participants available). Another approach might be to use data from past cohort studies where JA and maternal sensitivity have been adequately measured and screen the adult offspring for psychotic experiences and TD along with CD in parents.

A final approach would be to carry out a high-risk cohort study measuring CD (if possible during the antenatal period), mother-related covariates (e.g. verbal IQ, multiple

deprivation, depression, attachment style, ToM, etc.), dyad-related variables (e.g. maternal mind-mindedness and sensitivity, joint attention etc.), genetic risk measured molecularly (polygenic risk scores might be calculated to reduce genetic risk assessed from DNA to a single, continuous variable, see Iyegbe, Campbell, Butler, Ajnakina, & Sham, 2014), and specific developmental mechanisms as precursors of psychotic experiences (e.g. ToM, insecure attachment or dissociation, Bentall & Fernyhough, 2008; Bentall et al., 2014). With data from different time-points, researchers could look at temporal changes and at the impact of the different variables across time. By pursuing these strategies of research it should be possible to reformulate our understanding of psychosis as the outcome of abnormal developmental trajectories that are influenced by both social and biological factors.

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## **Chapter 7. Parental communication and psychosis: A meta-analysis**

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## 7.1 Abstract

**Background:** Parental communication deviance (CD) has long been suggested as a potential risk factor for the development of psychosis and thought disorder in genetically sensitive offspring. However, the findings of the studies on the prevalence of CD in parents of psychotic patients have never been submitted to quantitative synthesis.

**Method:** PsycINFO was searched from January 1959 to January 2012 for studies on the prevalence of CD in parents of psychotic patients. This search was supplemented with the results from a much larger systematic search (PsycINFO, PubMed, EMBASE, and Web of Science) on childhood trauma and psychosis.

**Results:** A total of 20 retrieved studies ( $n = 1753$  parents) yielded a pooled  $g$  of large magnitude (0.97; 95% CI [0.76; 1.18]) with a significant amount of heterogeneity ( $Q = 33.63$ ;  $p = .014$ ;  $I^2 = 46.47$ ). Subgroup and sensitivity analysis of methodological features (study's design, comparison group, diagnostic criteria, CD rating method, inter-rater reliability not reported, year of publication, and verbosity) and demographic characteristics (level of education or offspring's age) revealed that pooled effect size was stable and unlikely to have been affected by these features.

**Conclusion:** CD is highly prevalent in parents of psychotic offspring. This is discussed in the broader context of adoption and longitudinal studies that have reported a  $G \times E$  interaction in the development of psychosis and thought disorder. A potential developmental mechanism is suggested to explain how CD may affect the developing offspring. The importance of further studies on CD and its potential value as a clinical concept are discussed.

“Daughter: What did *you* mean by a conversation having an outline? Has this conversation had an outline?

Father: Oh, surely, yes. But we cannot see it yet because the conversation isn’t finished. You cannot ever see it while you’re in the middle of it” (Bateson, 1972, p. 42).

## 7.2 Introduction

Over the last decade, there has been a renewed interest in the role of the environment in the development of psychotic experiences (Bentall, 2003; van Os, Kenis, & Rutten, 2010; van Os, Krabbendam, Myin-Germeys, & Delespaul, 2005; Varese et al., 2012). It is becoming increasingly evident that a coherent scientific account of these experiences cannot be accomplished without the integration of environmental variables (Bentall & Fernyhough, 2008; Read, Bentall, & Fosse, 2009). Among these, quality of the family environment has long interested researchers (Bateson, Jackson, Haley, & Weakland, 1956; Lidz, Cornelison, Terry, & Fleck, 1958; Wynne, Ryckoff, Day, & Hirsch, 1958). Family research in psychosis had its peak during the 1970s and 1980s with the publication of studies looking at different aspects of family interaction (Doane, 1978). Since then, interest in this field has somehow declined. This sociohistorical shift can be explained in part by 2 factors. The first factor is related to the emergence of neurobiological framework as a dominant paradigm of research in the field of psychosis (Read et al., 2009b). The second factor is related to sensitivities surrounding this line of research, and worries that this may lead to the stigmatization of families (Read et al., 2004). It is our view that the family environment cannot be excluded as an important

focus for both research and clinical intervention (Lobban & Barrowclough, 2009).

Indeed, there is strong evidence that variables such as expressed emotion (Butzlaff & Hooley, 1998; Hooley, 2007), family rearing environment (Carter et al., 2002; Tienari et al., 2004), or family communication (Goldstein, 1987; Wahlberg et al., 2004) affect the course and development of psychosis.

One of the most researched family variables in the field of psychosis is parental communication deviance (CD; Tienari & Wahlberg, 2008). CD refers to a form of intrafamilial communication that is vague, fragmented, and contradictory and that compromises the development and sharing of meaning between the parent and the offspring, leading to the consequent breakdown in communication (Wynne, 1981). The concept has a multidimensional structure (Jones, 1977; Singer, Wynne, & Toohey, 1978) and its frequency and severity are continuously distributed with no clear cut-off point (Miklowitz & Stackman, 1992; Wynne, 1981). Examples can range from linguistic characteristics such as the use of *contorted and peculiar language*, e.g., “This man is *in the process in thinking of the process of being a doctor*” (Miklowitz et al., 1991, p. 166) and *ambiguous linguistic referents*, e.g., “Kid stuff that’s one thing, but *something else* is different too” (Velligan, 1985, p. 18) to problems at the level of pragmatics such as *nonverbal disruptive behaviour* (Singer & Wynne, 1966b). Other areas of research have examined variables that can be assumed to be related to CD such as *double-bind* statements (Bateson et al., 1956) or thinking problems in the parents of psychotic offspring (McConaghy, 1959). However, some of these concepts have not been rigorously researched and, in some cases, the underlying concept does not necessarily reflect CD (e.g., thought disorder; Johnston & Holzman, 1979; Wynne, 1984).

CD was initially developed and operationalized by Wynne and Singer (Singer & Wynne, 1963, 1965a, 1965b; Wynne et al., 1958; Wynne & Singer, 1963a, 1963b), who devised a scoring system for the Rorschach and the Thematic Apperception Test (Singer & Wynne, 1966b). Since then, the field has evolved with the development of new methodologies (Kymalainen, Weisman, Rosales, & Armesto, 2006; Velligan et al., 1990), prospective cohort designs (Goldstein, 1987; Wynne et al., 1987), and adoption studies that have helped elucidate the role of gene-environment interactions in determining the cross- generational transmission of psychotic communication disorders (Metsänen et al., 2005; Roisko et al., 2011; Siira et al., 2007; Wahlberg et al., 1997, 2000, 2004). The most important and replicated finding in the field is that CD is highly prevalent in the parents of psychotic offspring across diagnoses (Miklowitz & Stackman, 1992; Miklowitz et al., 1991). Some authors have, therefore, suggested that exposure to this kind of communication during childhood may play a causal role in the development and ontogeny of psychotic experiences (Wynne et al., 1977; Wynne, 1981, 1984). However, there is still no known mechanism by which CD may affect the offspring. Some researchers have shown that CD can be successfully measured during problem solving where parent and psychotic offspring are asked to discuss a salient family problem (Velligan et al., 1996, 1995, 1990). In other studies, it has been shown that parents with high scores on CD show less topic and affective focus during family discussions (Lewis et al., 1981; Lieber, 1977). Therefore, it is conceivable that the continuous exposure to communication that is vague, fragmented, and contradictory may lead the developing offspring to *internalize* it, resulting in psychotic experiences and thought disorder in particular. (Rund, 1985; Wynne, 1981). However, other mechanisms of transmission are

clearly possible.

Since Wynne and Singer's early work (Singer & Wynne, 1963, 1965a, 1965b; Wynne & Singer, 1963a, 1963b), the prevalence of CD among parents of psychotic offspring has been independently replicated by other groups using a variety of designs (Docherty et al., 1999; Docherty, 1993; Goldstein, 1987; Johnston & Holzman, 1979; Kymalainen et al., 2006; Morris & Wynne, 1965; Palombo et al., 1967; Wahlberg et al., 2004; Wild et al., 1975, 1965), with some studies documenting an exposure-response relationship between parental CD and severity of psychosis in the offspring (Docherty et al., 1999, 1997, 1994; Wynne et al., 1977). Other studies have shown more specific associations between CD and thought disorder (Sass et al., 1984), distractibility (Nuechterlein et al., 1989; Wagener et al., 1986), and relapse (Rund et al., 1995; Velligan et al., 1996). Moreover, CD does not seem to be a culture-bound phenomenon (Behrens et al., 1968; Carlson, 2011; Doane et al., 1989) and has been found to be relatively uninfluenced by parents' level of education (Docherty & Gordinier, 1999; Rund, 1986) or amount of speech produced (verbosity; Johnston & Holzman, 1979; Solana, 1988; Wynne et al., 1977).

Despite previous reviews (Hirsch & Leff, 1975; Jacob, 1975; Kymalainen & Weisman de Mamani, 2008; Liem, 1980; Miklowitz & Stackman, 1992), the literature in the field has never before been subjected to a quantitative analysis. Such analysis will allow us to establish the overall effect size for CD among parents of psychotic patients and determine its magnitude and consistency across studies. This analysis will also allow us to examine the impact of different study features that might affect our confidence in the findings. Finally, in conducting this analysis, we were also interested in comparing

effect sizes between mothers and fathers. According to Wynne and Singer, the presence of at least 1 parent with low CD should be a protective factor and hence that CD is required in both parents for psychosis to develop. If this is the case, we would expect the magnitude of the effect to be the same for both parents. If, on the other hand, there is a difference in the magnitude of the effects for mothers and fathers, this might point to moderating factors that affect the mechanism by which CD confers risk of psychosis.

## **7.3 Methods**

### **7.3.1 Literature search strategy and eligibility criteria**

Two of the authors (F.V. and R.P.B.) undertook an initial search of all the published and unpublished materials on CD as part of a more extensive meta-analysis on childhood experiences and psychosis. Specific details of the search strategy can be found elsewhere (Varese et al., 2012). This initial search yielded 47 studies that were screened for coding at the phase 4 of the present search. To check this first search, which focused on a wide range of potential environmental risk factors, a second complementary search was undertaken specifically focusing on CD and psychosis. This was limited to the time period between January 1959 and January 2012. The starting year was chosen because this was the date of the first empirical study published on thought disturbance in parents of patients diagnosed with schizophrenia (McConaghy, 1959). PsycINFO was searched using the following search terms: “communication deviance,” “communication disturbance\*,” and “thought dis\*” combined with the terms *famil\**, *parent\**, *mother*, *father* combined with *schiz\**, and *psycho\** using Boolean operator “and” and “or.” To minimize publication bias, we included unpublished material in our search and tracked



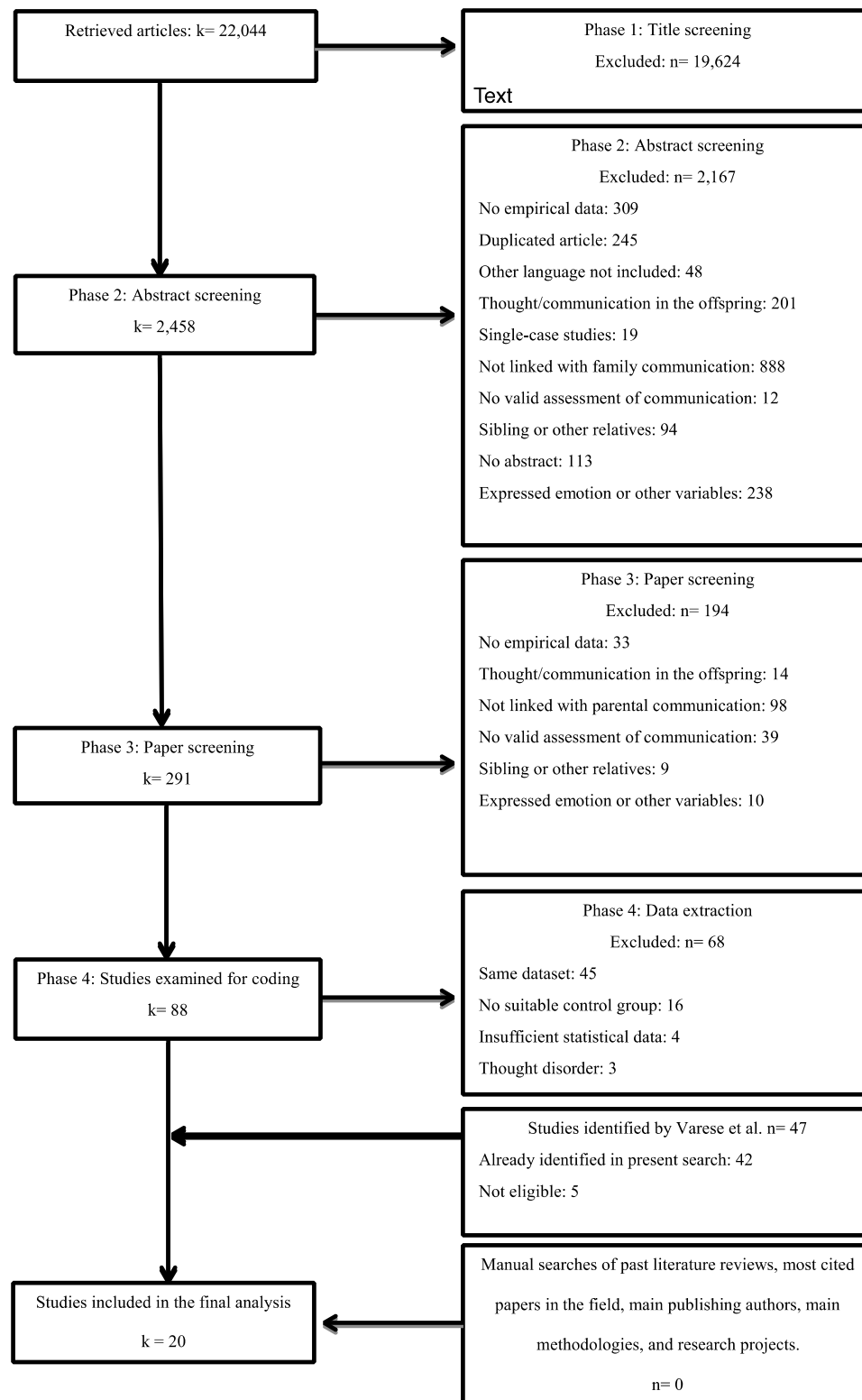
the citations of the most cited articles in the field (Singer & Wynne, 1965a, 1965b; Wynne & Singer, 1963a, 1963b). Concurrently, secondary searches were conducted using the names of the main publishing authors in the field, main methodologies, and research projects. Finally, we manually searched the bibliographic references of previous reviews in the field for material that had not been identified in our primary search (Hirsch & Leff, 1975; Miklowitz & Stackman, 1992) and contacted available authors in the field for further information about their published and unpublished work.

One exclusion criterion was the use of “artificial family” designs (Dell, 1977; Liem, 1974; Waxler, 1974). These designs involve experimental procedures in which family members of psychotic offspring interact with healthy offspring and parents of healthy offspring interact with psychotic offspring. These studies raised important issues about ecological validity and have used highly modified versions of Wynne and Singer’s methodology.

Also excluded were studies that operationalized *concept formation* using methodologies such as the object-sorting test (Catts et al., 1992; Lidz et al., 1962; McConaghy, 1959; Romney, 1969; Rosman, Wild, Ricci, Fleck, & Lidz, 1964; Schopler & Loftin, 1969a, 1969b). In these studies, communication is not tested through means of a speech sample. Furthermore, they have already been subject to a meta-analysis and review (Catts, Mcconaghy, Ward, Fox, & Hadzipavlovic, 1993). Studies that used analogue measures of CD such as consensus tasks (Loveland, Wynne, & Singer, 1963; Loveland, 1967; Shapiro & Wild, 1976; Singer, 1968) were also excluded. The dependent variable in these studies is the product of an interaction and so does not allow for a quantification of CD in parents individually. Another set of excluded studies were those

where the dependent variable was *disconfirmatory feedback* or *disqualifying communication* (Holte & Wichstrøm, 1990a, 1990b, 1991; Wichstrom et al., 1996; Wichstrøm et al., 1996; Wichstrøm, Holte, Husby, & Wynne, 1993; Wichstrøm & Holte, 1991, 1992). These constructs, despite measuring the quality of the family communication, only partly cover Wynne and Singer's original construct. The final exclusion criterion involved studies that have measured subclinical thought disorder in parents of psychotic offspring (Haimo & Holzman, 1979; Harrow & Quinlan, 1985; Johnston & Holzman, 1979) because CD and thought disorder are not necessarily correlated in such samples (Johnston & Holzman, 1979).

We initially retrieved a total of 22044 titles. Figure 10 shows the 4 phases of the systematic search. Only 20 studies were found to be eligible for analysis. In part, this was due to the fact that many of the studies retrieved were based on the same data set (Docherty et al., 1999; Docherty & Gordinier, 1999; Docherty, Hall, & Gordinier, 1998b; Docherty, Rhinewine, et al., 1998) or were part of the same research project such as the *UCLA High-Risk Project* (Albers et al., 1986; Doane et al., 1981; Goldstein, 1987; Lewis et al., 1981; Lieber, 1977). In cases where studies had been based on the same data set, we selected the articles with the most complete statistical information for effect size computation.



**Figure 10** - Flowchart of studies included in the meta-analysis.

Source	Study type	Parents (n)	Controls (n)	Control groups	Education (Y/N)	Diagnostic criteria ( $\geq$ DSM-III)	Age of the offspring (>15)	Method	Scoring	Parent	IRR (Y/N)	Verb. (Y/N)
Asarnow et al. (1988)	CCS	28	72	Mixed	Y	Y	N	Projective	CD	Mother	Y	Y
Behrens et al. (1968)	CCS	56	22	Healthy	Y	N	Y	Projective	CD	Both	Y	N
Docherty & Gordinier (1999)	CCS	59	24	Healthy	Y	Y	Y	Other	CDI	Both	Y	Y
Docherty (1993)	CCS	18	10	Healthy	Y	Y	Y	Other	ICD	Both	Y	Y
Glaser (1976)	CCS	20	20	Mixed	Y	N	Y	Projective	CD	Both	Y	Y
Goldstein, (1987)	PCS	128		N/a	Y	Y	Y	Projective	CD	Both	Y	Y
Hirsch & Leff (1971)	CCS	40	40	Other	Y	N	Y	Projective	CD	Both	Y	Y
Holte & Wichstrøm (1990)	CCS	14	28	Mixed	Y	Y	Y	Projective	EU	Both	Y	Y
Johnston & Holzman (1979)	CCS	24	34	Mixed	Y	N	Y	Projective	CD	Both	Y	Y
Jones (1977)	CCS	15	12	Mixed	Y	N	Y	Projective	CD	Both	Y	Y
Rund (1986)	CCS	42	58	Mixed	Y	Y	Y	Projective	CD	Both	Y	Y

Sass et al. (1984)	CCS	42	8	Other	Y	Y	Y	Projective	CD	Both	Y	Y
Singer & Wynne (1963)	CCS	40	80	Other	N	N	N	Projective	CD	Both	N	N
Singer et al. (1978)	CCS	26	16	Other	N/K	N	Y	Projective	CD	Both	N	Y
Solana (1988)	CCS	40	40	Other	Y	Y	Y	Projective	CD	Both	Y	Y
Wender et al. (1977)	CCS	56	28	Other	N	N	Y	Projective	CD	Both	Y	Y
Wild et al. (1965)	CCS	44	49	Healthy	Y	N	Y	Other	CD	Both	N	N
Wild et al. (1975)	CCS	72	102	Mixed	Y	N	Y	Other	CD	Both	Y	Y
Wynne (1967)	CCS	38	80	Mixed	N	N	Y	Projective	CD	Both	N	N
Wynne et al. (1977)	CCS	88	140	Mixed	Y	N	Y	Projective	CD	Both	Y	Y

**Note:** CCS, case-control study; CD, communication deviance; CDI, Cognitive Disturbances Index; DSM-III, Diagnostic and Statistical Manual of Mental Disorders-III; EU, egocentric utterances; healthy, parents of healthy offspring; ICD, interactional communication deviance; IRR, inter-rater reliability reported; Verb, verbosity accounted for (e.g. word count); mixed, multiple contrast groups; N, no; N/a, not applicable; N/K, not known; other, parents of patients with other mental health diagnoses; PCS, prospective cohort study; projective, Rorschach, TAT, or Phillipson; Y, yes.

**Table 18** - Characteristics of the studies included in the meta-analysis.

### 7.3.2 Coding protocol and effect sizes (g) computation

The primary goal of the protocol was to allow a detailed subgroup analysis based on the methodological features that were likely to influence effect sizes. The following study characteristics were coded (see table 18): age of the offspring, diagnostic criteria, control group, type of methodology, and education or verbosity accounted for.

The computation of effect sizes and consequent statistical analysis were performed using Comprehensive Meta-analysis (Biostat, 2005). This software allows the user to easily compute effect sizes using a wide variety of data formats.

The computation of effect sizes was performed using *hedges' g* (Hedges, 1981) given that CD is a continuous construct with no real threshold values. In some studies, the means, SDs, samples sizes, and *p* values for both the experimental and control groups were available (Docherty et al., 1999; Wynne et al., 1977) and therefore *g* was calculated using the original equation (Figure 11).

$$g = \frac{Me - Mc}{SD \text{ pooled}} \times \left( \frac{N - 3}{N - 2.25} \right) \times \sqrt{\frac{N - 2}{N}}$$

**Figure 11** – Hedges' *g* formula.

Here, *Me* stands for mean of the experimental group, *Mc* for mean of the control group, and *N* for number of participants. In 2 cases (Sass et al., 1984; Singer et al., 1978), SDs were not available and *g* was calculated using the means, sample sizes, and *p* values for both groups of parents. In 1 case (Solana, 1988), *g* was computed from *t* test, *p* value, and effect direction given that no other statistical data were available.

In studies where the dependent variable was dichotomous (Asarnow, Goldstein, & Benmeir, 1988; Behrens et al., 1968; Goldstein, 1987; Jones, 1977; Singer & Wynne, 1963; Wender, Rosenthal, Rainer, Greenhill, & Sarlin, 1977; Wild et al., 1975, 1965), standard means difference (SMD) was calculated from OR using agreed statistical conventions (Figure 12; Chinn, 2000; Higgins & Green, 2011).

$$\text{SMD} = \frac{\sqrt{3}}{\pi} \ln \text{OR}$$

**Figure 12** - Convention to calculate standard means difference from odds ratio.

Finally, in one study,  $g$  was calculated from the chi-square ( $\chi^2$ ),  $p$  value, and sample size (Wynne, 1967) given that no other statistical information was available. In studies where the research design included more than 2 groups, the effect size was calculated from the comparison between parents of offspring diagnosed with schizophrenia and parents of healthy offspring (Johnston & Holzman, 1979; Rund, 1986; Wynne et al., 1977). For studies that had used more than 1 concept of CD, only the data that reflected Wynne and Singer's original conceptualization were extracted.

Finally, computation of effect sizes was carried out under random effects model given that our assumption was that the studies retrieved were likely to be heterogeneous and the analysis was likely to carry across-study variation (Hedges, 1981).

## 7.4 Results

### 7.4.1 Pooled effect sizes and heterogeneity analysis

The final analysis included 19 case-control studies and data from 1 prospective study (Goldstein, 1987). The studies included an overall pooled sample of 1753 parents. The computation carried out under a random effects model for the entire sample revealed a very large pooled effect size of 1.45 (SE = 0.27; 95% CI [0.92; 1.97];  $z = 5.41$ ;  $p < .001$ ), with a significant amount of heterogeneity ( $Q[19] = 238.8$ ;  $p < .001$ ;  $I^2 = 92.04$ ). After visual inspection of the funnel plot, we decided to exclude Wynne et al. (1977) because this effect size was of an unusually large magnitude ( $g = 12.4$ ; SE = 0.8; 95% CI [10.84; 13.97];  $z = 15.54$ ;  $p < .001$ ). The exclusion of this outlier reduced the overall effect size to 0.97 (SE = 0.11; 95% CI [0.76; 1.18];  $z = 9.2$ ;  $p < .001$ ), with a more acceptable, but still significant, level of heterogeneity ( $Q[18] = 33.63$ ;  $p = .014$ ;  $I^2 = 46.47$ ;  $\tau^2 = 0.1$ ;  $\tau = 0.3$ ). According to benchmark thresholds (Cohen, 1988b), we can interpret that our pooled  $g$  is of large magnitude. One-study removed analysis revealed that the results were stable and unlikely to be affected by the exclusion of any one study.

In order to test how the different features affected our result, we recomputed the pooled effect size extracting studies using our coding protocol. The exclusion of 1 cohort study did not change our overall effect size ( $k = 18$ ;  $g = 0.96$ ; SE = 0.11; 95% CI [0.75; 1.16];  $z = 8.95$ ;  $p < .001$ ;  $Q[17] = 32.42$ ;  $p = .013$ ;  $I^2 = 47.56$ ;  $\tau^2 = 0.09$ ;  $\tau = 0.3$ ). The computation of the effect size using studies that had compared parents of psychotic offspring with healthy controls (as opposed to other kinds of controls, e.g., parents of children with depression or learning difficulties), again, did not change the pooled effect size significantly ( $k = 5$ ;  $g = 0.91$ ; SE = 0.24; 95% CI [0.44; 1.38];  $z = 3.79$ ;  $p < .001$ ;



$Q[4] = 14.76; p = .005; I^2 = 72.91; \tau^2 = 0.2; \tau = 0.45$ ). However, heterogeneity increased significantly and the CI broadened. The exclusion of studies that had not controlled for the parents' educational level brought the effect size to a still significant and large 0.89 ( $k = 15; SE = 0.11; 95\% CI [0.67; 1.1]; z = 7.97; p < .001; Q[14] = 22.29; p = .073; I^2 = 37.18; \tau^2 = 0.06; \tau = 0.25$ ). The exclusion of studies that had tested parents of children below the age of 15 again did not change the overall pooled effect size ( $k = 17; g = 0.94; SE = 0.1; 95\% CI [0.73; 1.14]; z = 8.99; p < .001; Q[16] = 27.39; p = .037; I^2 = 41.59; \tau^2 = 0.07; \tau = 0.27$ ). Unfortunately, we could not carry out a subgroup analysis by offspring's sex because there were too few data. Finally, we recomputed the effect size for studies that had accounted for verbosity. This reanalysis brought the pooled  $g$  down to a still large and significant 0.83 ( $k = 15; SE = 0.11; 95\% CI [0.63; 1.04]; z = 7.93; p < .001; Q[14] = 20.09; p = .127; I^2 = 30.32; \tau^2 = 0.05; \tau = 0.22$ ), whereas the effect size for studies that did not account for verbosity was 1.35 ( $k = 4; SE = 0.16; 95\% CI [1.03; 1.66]; z = 8.42; p < .001; Q[3] = 3.31; p = .346; I^2 = 9.45; \tau^2 = 0.01; \tau = 0.1$ ) and difference between the 2 types of study was significant ( $Q[1] = 7.22; p = .007$ ).

Because diagnostic criteria have changed considerably since Wynne and Singer's early studies, we decided to run a subgroup analysis of the studies carried out before and after the publication of Diagnostic and Statistical Manual of Mental Disorders-III (DSM-III). The estimated effect size for the latter studies was 0.96 ( $k = 8; SE = 0.2; 95\% CI [0.57; 1.36]; z = 4.78; p < .001$ ), however, with a significant level of heterogeneity ( $Q[7] = 16.75; p = .019; I^2 = 58.22; \tau^2 = 0.18; \tau = 0.42$ ). The pooled effect size for the studies undertaken before the publication of the DSM-III was 0.98 ( $k = 11; SE = 0.12; 95\% CI [0.74; 1.22]; z = 8.02; p < .001$ ), but with a non-significant level of heterogeneity ( $Q[10]$

= 16.37;  $p = .09$ ;  $I^2 = 38.9$ ). To complement the above analysis, we carried out a meta-regression using year of publication as a moderator variable. The regression was carried out using mixed effects to allow for between-study heterogeneity. Overall, year of publication was not found to be a significant predictor of effect size ( $\beta = -0.01$ ; SE = 0.01; 95% CI [-0.03; 0.02];  $z = -0.51$ ;  $p = .61$ ;  $\alpha = 11.68$ ; SE = 20.99; 95% CI [-29.47; 52.82];  $z = 0.56$ ;  $p = .58$ ;  $\tau^2 = 0.1$ ;  $Q_R = 0.26$ ;  $p = .61$ ;  $Q_E = 14.82$ ;  $p = .61$ ;  $Q_T = 15.08$ ;  $p = .66$ ).

Another potential source of heterogeneity between studies was the diverse methodologies used to elicit speech samples. A subgroup analysis of the studies that have used projective techniques revealed a  $g = 0.93$  ( $k = 16$ ; SE = 0.13; 95% CI [0.68; 1.18];  $z = 7.17$ ;  $p < .001$ ) with a significant amount of heterogeneity ( $Q[15] = 28.49$ ;  $p = .012$ ;  $I^2 = 50.87$ ;  $\tau^2 = 0.12$ ;  $\tau = 0.34$ ), whereas studies that have used other methodologies yielded a  $g$  of 1.09 ( $k = 4$ ; SE = 0.15; 95% CI [0.79; 1.39];  $z = 7.05$ ;  $p < .001$ ) with a non-significant level heterogeneity ( $Q[3] = 3.45$ ;  $p = .327$ ;  $I^2 = 13.04$ ).

We were also interested in the effect sizes per parental sex. The effect size for mothers of psychotic offspring was  $g = 0.89$  ( $k = 7$ ; SE = 0.18; 95% CI [0.54; 1.24];  $z = 4.99$ ;  $p < .001$ ;  $Q[6] = 7.92$ ;  $p = .244$ ;  $I^2 = 24.21$ ). Analysis of the effect sizes for the fathers of psychotic offspring revealed a much smaller  $g = 0.39$  ( $k = 6$ ; SE = 0.16; 95% CI [0.07; 0.7];  $z = 2.42$ ;  $p < .05$ ;  $Q[5] = 5.06$ ;  $p = .41$ ;  $I^2 = 1.2$ ). Using a mixed effect analysis, the comparison revealed that the difference between the 2 mean effect sizes was statistically significant ( $Q[1] = 4.38$ ;  $p < .05$ ).

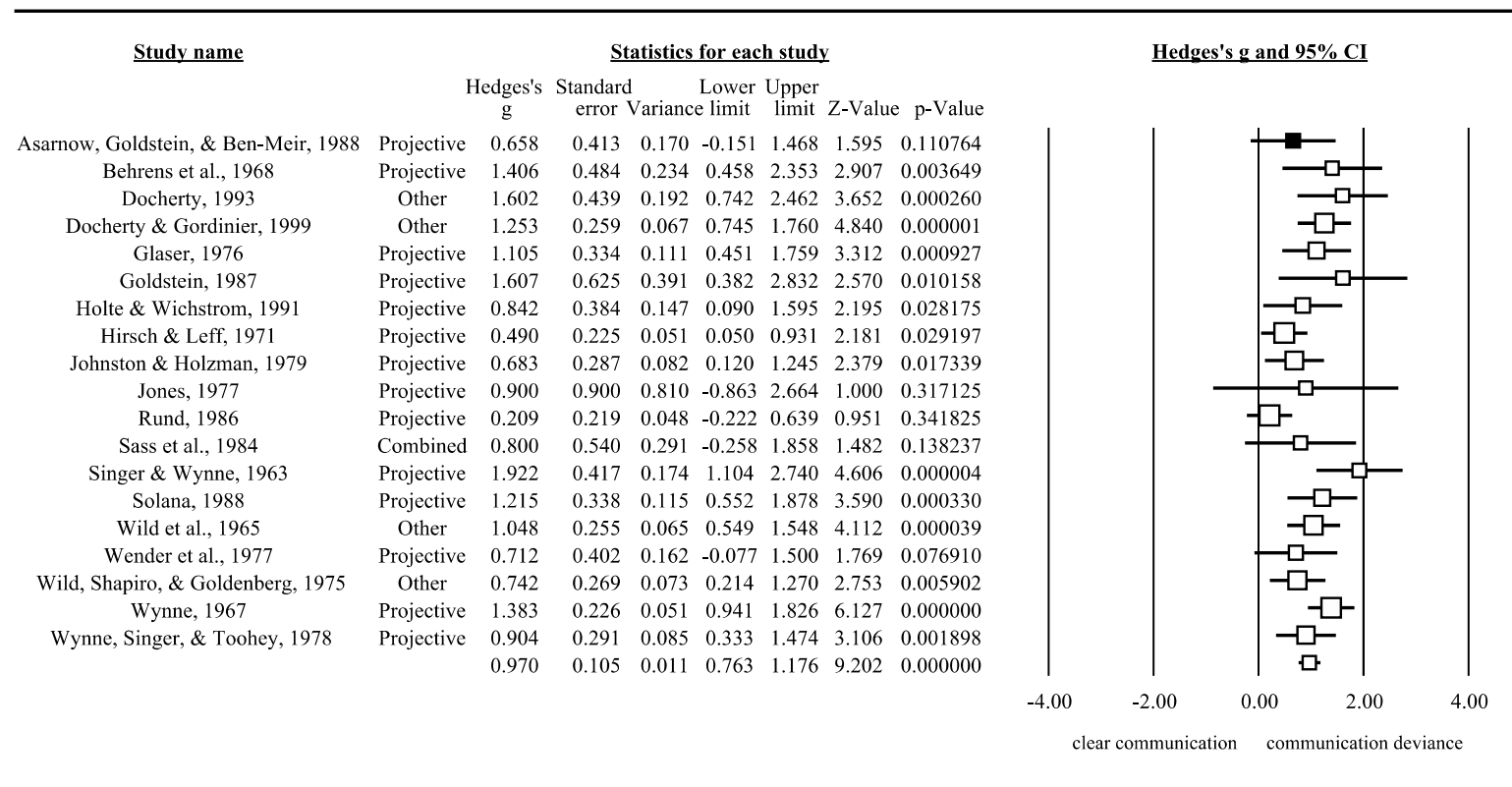
Finally, we ran subgroup analysis between studies that have reported inter-rater reliability (IRR) ( $k = 14$ ;  $g = 0.87$ ; SE = 0.12; 95% CI [0.64; 1.11];  $z = 7.25$ ;  $p < .001$ ;

$Q[13] = 21.49; p = .064; I^2 = 39.5; \tau^2 = 0.07; \tau = 0.27$ ) and studies that either had not reported IRR or in which reliability was poor (e.g. Wender et al., 1977) ( $k = 5; g = 1.18; SE = 0.17; 95\% CI [0.84; 1.52]; z = 6.83; p < .001; Q[4] = 6.5; p = .165; I^2 = 38.45; \tau^2 = 0.06; \tau = 0.24$ ). Using a mixed effect analysis, the comparison revealed that the difference between the 2 mean effect sizes was not statistically significant ( $Q[1] = 2.1; p = .147$ ).

#### **7.4.2 Publication bias**

The visual inspection of the funnel plot revealed potential publication bias. The following statistical tools were used to explore this: (1) Begg and Mazumdar's rank order correlation, (2) Egger's regression intercept, and (3) Duval and Tweedie's "trim and fill" procedure. Begg and Mazumdar's test (Begg & Mazumdar, 1994) revealed a non-significant Kendall's  $\tau$  of 0.22 ( $z = 1.33; p = .09$ ). Although this non-significant value suggests nonexistence of publication bias, the test has relatively low power for meta-analyses with a small number of studies.

In Egger's test (Egger, Davey Smith, Schneider, & Minder, 1997), funnel plot asymmetry was calculated from a linear regression where the more the intercept deviates from 0, the more pronounced the asymmetry. Given the small number of pooled studies, evidence for asymmetry was taken at  $p < .1$ . In our case, the intercept was 1.49 (95% CI  $[-0.54; 3.52]; t[17] = 1.55; p = .07$ ) supporting the existence of bias. Finally, Duval and Tweedie's "trim and fill" procedure (Duval & Tweedie, 2000) identified 2 potential missing studies. The recomputed point estimate was 0.91 (95% CI  $[0.7; 1.13]$ ) revealing that the presence of these "missing studies" was not likely to affect the overall magnitude of our effect size.



**Figure 13 - Forest plot.**

## 7.5 Discussion

The overall pooled effect size of the studies we considered was large in magnitude supporting Wynne and Singer's clinical intuitions and early findings. This effect was observed to be larger for mothers than for fathers.

It can be argued that these results should be treated cautiously given that there was considerable heterogeneity between studies. However, we have tried to circumvent this problem by combining individual effect sizes using a random effects model by excluding an outlier and finally through subgroup and sensitivity analyses, which brought down the heterogeneity to more acceptable levels. The most striking consequence was that all of these analyses continued to reveal large and significant effect sizes suggesting that variation between studies should not undermine our overall confidence in the association between CD in parents and psychosis in offspring. It is important to note, however, that the result of Egger's test suggested the existence of publication bias. When we recomputed a point estimate using the "trim and fill" procedure, we found that potential missing studies were not likely to undermine the presence of a significant effect. However, the existence of publication bias is an important issue that cannot be disregarded completely.

Some between-study variability should be unsurprising given the many limitations that these studies have in terms of their methodological quality. For example, many of the studies that were undertaken before the publication of DSM-III used diagnostic approaches that were quite different from today's standardised methods. On this issue, it was reassuring to see that a subgroup analysis, based on this specific methodological variable, yielded stable effect sizes across groups. Also, meta-regression

analysis showed that year of publication of the study had no impact on overall effect size. A second problem affecting some of the old studies is the multiplicity of different methodologies used to gather speech samples. This plurality has the advantage of showing that effect sizes are not an artefact of any specific methodology. However, it makes the task of quantitative synthesis difficult and is likely to be a source of significant amount of heterogeneity. This is especially relevant when one looks at the differences in the methods of standardisation of the CD measures. For example, in some studies, this ratio was calculated from word counts (Hirsch & Leff, 1971), whereas in others studies, it was calculated using the number of lines of speech (Docherty, 1993). On this specific limitation, it was reassuring to note that the magnitude of the effect size did not change when we sub-analysed the results using methodology as a criterion. It is also interesting that some studies have replicated Wynne and Singer's early findings using sophisticated and standardised linguistic methodologies (Docherty et al., 1999) showing that findings on CD can be replicated using more natural conversational samples.

Another issue that limited the power of the present meta-analysis is the lack of standardised threshold values. In the studies in which results were reported as dichotomous outcomes, we have opted to reconstruct a continuous variable using agreed statistical conventions. However, we are aware that this approach has some limitations. In this respect, we felt reassured by the fact that nearly all the studies identified and retrieved reported positive findings and by the fact that our analysis comparing dichotomous and continuous outcomes revealed a stable effect size.

Another limitation is related to issues of reliability and validity. If it is true that in the majority of studies IRR has been ascertained and reported, it is no less true that in a few

studies this issue, if not completely ignored, at least was not discussed and reported. Our subgroup analysis revealed that differences in mean effect sizes between studies that reported IRR and studies that did not were not significant.

Finally, our analysis tells us very little about the explanation for the prevalence of CD in parents of psychotic offspring. Given the robustness of the association between psychosis in offspring and parental CD, it is pertinent to consider the possible processes that might account for it.

Speculating about reverse causality, some authors have suggested that the higher prevalence of CD in parents of psychotic offspring could reflect a reaction of the parent to the disordered communication of the offspring (Covelman, 1977; Dell, 1977; Liem, 1974, 1976, 1980; Waxler, 1974). However, rigorous studies have shown that parents' CD measured during individual protocols correlates positively with CD during problem-solving situations with their offspring (Velligan et al., 1990), suggesting that variance in CD is not explained by the offspring's immediate behaviour (Glaser, 1976; Roisko et al., 2011).

In an attempt to settle the issue, prospective and adoption studies have demonstrated that CD in the parent precedes the development of psychosis in the offspring by many years (Goldstein, 1987; Wahlberg et al., 2004), and more importantly, that healthy communication in an adopting couple seems to exert a protective effect in the case of high-risk adoptees (Wahlberg et al., 1997). Also relevant to the question of reverse causality is the observation that CD has the quality of an enduring trait-like characteristic in parents (Nugter et al., 1997; Rund et al., 1995; Velligan et al., 1995) that becomes stable in the transition from adolescence to adulthood (Wahlberg et al., 2001)

and that does not worsen with arousal or stress in the parent (Docherty, Hall, et al., 1998b; Docherty et al., 1994). These studies as a whole support the view that CD, rather than a reactive and transient phenomenon (Schopler & Loftin, 1969b), seems to be an enduring characteristic of some parents (Nugter et al., 1997; Rund et al., 1995). Despite these observations, it remains possible that CD may become involved in a complex dynamic process of circular causality where cause and effect are intertwined (Read et al., 2004; Wichstrøm & Holte, 1992) as appears to be the case with other family variables such as *expressed emotion* (Hahlweg et al., 1989; Miklowitz, 1994; Wearden, Tarrier, Barrowclough, Zastowny, & Rahill, 2000).

Another hypothesis that has been suggested to explain the prevalence of CD among parents of psychotic offspring is that this form of communication could be an epiphenomenon of shared genetic vulnerability to psychosis, i.e., *endophenotype* (Kinney et al., 1997; Shenton, Solovay, Holzman, Coleman, & Gale, 1989; Wender et al., 1977). According to this account, CD among parents of psychotic offspring should be interpreted against a broader context of cognitive deficits that are believed to be an expression of genetic liability for schizophrenia among unaffected first-degree relatives (Snitz, MacDonald, & Carter, 2006).

Some researchers have suggested that the *FOXP<sub>2</sub>* gene (*CNTNAP<sub>2</sub>* pathway) could be responsible for a shared vulnerability to CD and thought disorder (Levy et al., 2010; Tolosa et al., 2010) although a recent meta-analysis of 2 genome-wide association studies found a significant association between thought disorder and 4 other genetic loci (*PKNOX<sub>2</sub>*, *MYH<sub>13</sub>*, *PHF<sub>2</sub>*, and *GPC<sub>6</sub>*; Wang, Zhang, Liu, Wu, & Zeng, 2012). However, an exclusively genetic account seems unlikely given that methodologically rigorous



studies have shown that CD is a transdiagnostic risk factor for psychiatric disorders other than schizophrenia (Hamilton, Jones, et al., 1993; Wynne et al., 1976). Furthermore, in our meta-analysis, a larger effect was found for maternal CD than for paternal CD.

Although sex-linked genetic effects are not impossible, an obvious explanation for this finding is that actual exposure to CD is required for there to be an increased risk in the offspring. This sex effect appears to suggest that low CD in one of the parents (fathers) by itself may not have the protective impact on the development of psychosis that Wynne and Singer initially hypothesized. However, we were not able to extract data comparing families in which both parents had CD vs. families in which only 1 parent had CD, and we therefore could not test the protection hypothesis directly.

Interestingly, evidence of a gene-environment interaction was reported by Tienari and colleagues in a relatively small study (Wahlberg et al., 1997, 2000, 2004), in which it was found that at-risk children only developed a psychosis and thought disorder if they were raised by adoptive parents who exhibited CD. This finding has never been replicated and, if found to be secure in further studies, would provide one of the few examples of gene-environment interactions known to be important in psychosis. Hence, an important avenue for future research might be to measure parental CD alongside genetic data obtained from offspring in studies comparing patients and controls.

Prospective cohort studies could help us clarify the mechanism by which exposure to CD may affect the development of psychosis (Wahlberg et al., 2004) and thought disorder (Wahlberg et al., 1997, 2000) in offspring, which might ultimately have important implications for the prevention of psychosis and thought disorder. Miklowitz and Stackman (1992) have suggested that exposure to CD might act as a psychosocial

stressor that particularly affects genetically sensitive children. If this is the case, it might be expected that CD will affect the likelihood of a wide range of psychotic and affective symptoms. An alternative possibility is that CD affects some symptoms more than others. Indeed, some studies have focused specifically on the relationship between parental CD and thought disorder in offspring (e.g. Sass et al., 1984) although, to our knowledge, none have carried out adequate statistical controls for the co-occurrence of symptoms. Since Wynne and Singer first began their work on CD, more has been discovered about the structure of psychosis and its course across time. Although more complex models have also been proposed (Reininghaus et al., 2012), a widely supported model proposes that all psychotic disorders can be described along 5 symptom dimensions: positive symptoms, negative symptoms, cognitive disorganisation, depression, and mania (Demjaha et al., 2009). Future research should consider whether CD is specifically related to any of these dimensions.

Consistent with the idea that actual exposure to CD is required to increase the risk of psychosis, some authors have argued that the impact of maternal CD is likely to occur during early development through the progressive internalization of language during social interaction with the parent (Rund, 1985; Velligan et al., 1988; Wynne, 1981, 1984), a hypothesis that is consistent with Vygotsky's sociocultural analysis of cognitive development (Vygotsky, 1934). Another hypothesis is that the inability to establish and maintain shared foci of attention in the parent has a specific effect on very early non-verbal reciprocal dialogues between mother and baby, leading to the disruption of early cognitive development and communication in the child, resulting in a high risk of psychosis (Wynne, 1968). Interestingly, and despite remaining completely unexplored,

this hypothesis is consistent with work by researchers in the field of developmental psychology, whose studies have demonstrated the crucial role of the mother in scaffolding the child's cognitive development during early episodes of joint attention (Carpenter et al., 1998; Goldsmith & Rogoff, 1997b; Hobson et al., 2004; Hustedt & Raver, 2002; Miller, Ables, King, & West, 2009; Mundy & Jarrold, 2010; Scaife & Bruner, 1975; Tasker & Schmidt, 2008; Tomasello & Farrar, 1986). Along with other ante- and perinatal risk factors that have been suggested within the framework of the neurodevelopmental hypothesis of schizophrenia, such a developmental pathway could in part explain the results of birth cohort studies that have documented early developmental delays in children who were later diagnosed with schizophrenia (Bearden et al., 2000; Cannon et al., 2002; Jones, Rodgers, Murray, & Marmot, 1994). This hypothesis is also consistent with studies that show an association between maternal CD and poor cognition in offspring diagnosed with schizophrenia (Nuechterlein et al., 1989; Rund, 1985; Wagener et al., 1986) and with our present finding of a lower prevalence of CD in fathers of psychotic offspring. This hypothesis should be considered in future cohort studies.

At a clinical level, the revival of research on CD may have important implications for the development of family interventions. Results from a systematic review (Bird et al., 2010) and meta-analyses (Pharoah, Mari, Rathbone, & Wong, 2006; Pilling et al., 2002) have clearly shown that these interventions are effective in reducing psychotic relapse and hospital admissions. Parental CD has also been found to be associated with psychotic relapse in the offspring in 2 independent studies (Rund et al., 1995; Velligan et al., 1996). Hence, a fruitful avenue for enhancing these interventions might be to target the quality of the family communication from a CD standpoint.

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**Chapter 8. Mapping early environment using  
communication deviance: A longitudinal study of  
maternal sensitivity towards 6-month-old children**

This paper is currently in preparation as de Sousa, P., Fien, K., Sellwood, W., Sharp, H., Hill, J., Pickles, A. & Bentall, R. P. (in preparation). Mapping early environment using communication deviance: A longitudinal study of maternal sensitivity towards 6-month-old children.



## 8.1 Introduction

### 8.1.1 Communication Deviance

The concept of communication deviance (CD) was generated in the context of attempts to account for environmental contributions to the development of schizophrenia (de Sousa, Varese, Sellwood, & Bentall, 2013). CD refers to qualities of communication that would leave a listener uncertain, puzzled and unable to share a focus of attention with the speaker. The construct reflects a range of perceptual-cognitive and linguistic-verbal reasoning disturbances that are believed to affect the establishment and maintenance of focus of attention during communication and hence compromise the development of conversational alignment and shared meaning between interlocutors (Miklowitz & Stackman, 1992; Nuechterlein, Goldstein, Ventura, Dawson, & Doane, 1989; Singer & Wynne, 1965a, 1965b; Wynne, Singer, Bartko, & Toohey, 1977; Wynne & Singer, 1963a, 1963b). These disturbances are subtle and can range from *ambiguous linguistic references* e.g. “Kid stuff that's one thing but something else is different too” (Velligan, et al., 1990) or contradictions e.g. “I didn’t get much sleep last night (interviewer: are you tired?) Yeah, I ain’t tired” (Docherty, 1993) to derogatory, disparaging or critical remarks or even more overarching non-verbal disturbances at the level of the pragmatics of communication e.g. mistimed turn-taking (Wynne et al., 1977).

According to the authors of the construct, Lyman Wynne and Margaret Singer, CD may contribute to the development of symptoms through its pervasive effect on the offspring’s socio-cognitive development during formative years (Wynne et al., 1977). They suggested that children learn to share and sustain foci of attention, and consequently derive meaning from the world around them, through communication with their parents

(Wynne, 1981, 1984). It was initially hypothesized that CD in the caregiver, in interaction with genetic vulnerability in the offspring, would lead to the escalation of cognitive and affective disturbances seen in psychosis (Wynne, 1981). Consistent with this hypothesis, Wahlberg and colleagues (1997, 2000) reported that the interaction between offspring's genetic risk and adoptive caregivers' CD were significant predictors of thought disorder in the adoptee. However, it is not yet clear whether parental CD is a risk factor that is specific for schizophrenia (Roisko, Wahlberg, Hakko, & Tienari, 2014). Indeed, it is possible that CD may reflect an important environmental risk for a range of mental health disorders (Wahlberg et al., 2004).

Evidence for the role of CD in relation to childhood disorders is limited. Cross sectional studies have found that parental CD in the mother is associated with poorer social, cognitive and emotional development in high-risk 7 and 10 year olds offspring of parents diagnosed with severe mental health disorders (Doane et al., 1982), and with social withdrawal and behavioural problems in 9 year olds (Velligan, Christensen, Goldstein, & Margolin, 1988). Findings from longitudinal studies using measures based on concepts allied to CD have been consistent with there being early effects. Disrupted caregiver-infant communication (e.g. caregiver's contradictory affective cues or withdrawal, Lyons- Ruth, Bronfman, 1999) at the age of 18 months has been shown to be a significant predictor of dissociative symptoms measured 20 years later (Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009).

Drawing from data collected in a high-risk prospective study (University of Rochester Child and Family Study, Wynne, Cole, & Perkins, 1987), other researchers have reported associations between parental communication that is vague, contradictory

and disconfirming and anxiety (Wichstrøm, Holte, & Wynne, 1993) and poorer social competence in 7 and 10 year old offspring (Wichstrøm, Holte, Husbey, & Wynne, 1994; Wichstrøm, Holte, Husby, & Wynne, 1993). Interestingly, in the same high-risk cohort, but at longer follow-up (offspring were at least 18 years of age), initial scores for disqualifying communication in parents were a significant predictor of psychological distress and well-being and global mental health in the offspring (Wichstrøm, Anderson, Holte, & Wynne, 1996) and disconfirmatory communication was a powerful predictor of poor interpersonal functioning and hospitalization for psychiatric disorders in the offspring (Wichstrøm, Anderson, Holte, Husby, & Wynne, 1996).

Wynne's original proposal was that child socio-cognitive development occurred along different domains of family *relatedness* such as caregiving, problem solving, mutuality and intimacy. These increasingly sophisticated relational domains represented different and evolving levels of family interaction (Wynne, 1984, 1988). Specifically, disturbances at the level of communication in the caregiver were hypothesized to have an expression at the more basic level of *relatedness* between the caregiver and the infant during early pre-verbal dialogues, disrupting early development (Wynne, 1968). In other words, caregiving was assumed to have a crucial communicative component and CD was conceptualized as a risk marker for parental mental processes that might give rise to disruptions to the caregiving system (Singer & Wynne, 1966). For example CD may reflect a general limitation in a parent's capacity to generate a reciprocal intersubjective space with the child, as a basis for shared meaning that will be evident in observed early parent-infant interactions prior to language. Despite Wynne and Singer's developmental account, this has not been examined previously. Furthermore, most studies of CD are

open to the criticism that associations with child behaviours may arise from evocative effects of children on parental *speech* (Miklowitz & Stackman, 1992), and associations between CD and parental *behaviours*, studied in cross-section, could arise from effects of children on both. In this study we assessed maternal CD during pregnancy, and maternal sensitivity following the birth of the infant, and so were able to ask whether there is a temporal association between CD and maternal sensitivity that could not be accounted for by infant behaviours.

### **8.1.2 Maternal sensitivity**

Maternal sensitivity, refers to the extent to which parental responses to infant cues are contingent, appropriate, interested and warm (Bornstein & Tamis-Lemonda, 1997). The importance of maternal sensitivity during infancy is supported by diverse findings including that low maternal sensitivity during infancy predicts harsh parental discipline during toddlerhood (Joosen, Mesman, Bakermans-Kranenburg, & van Ijzendoorn, 2012), and that maternal sensitivity interacts with MAOA polymorphisms to predict temperamental anger proneness (Pickles et al., 2013), and with DRD4 polymorphisms to predict child externalising behaviours (Bakermans-Kranenburg & Van Ijzendoorn, 2006). Associations between maternal sensitivity during infancy and academic and social functioning in adolescence (Fraley, Roisman, & Haltigan, 2012) and up to age 32 years (Raby, Roisman, Fraley, & Simpson, 2014) have been reported, suggesting an enduring effect. Fraley and colleagues took advantage of repeated measurements of maternal sensitivity and of social and academic competence over childhood, together with measures of potential confounders (in the NICHD Study of Early Child Care and Youth

Development) to examine whether there was an enduring independent contribution of maternal sensitivity to adolescent functioning (Fraley, Roisman, Booth-LaForce, Owen, & Holland, 2013). They found that the strength of association between maternal sensitivity and later social and cognitive functioning did not attenuate over time, and that it could not be accounted for by potential confounding variables such as maternal education, nor by maternal sensitivity over the intervening period between infancy and adolescence, nor by transactional processes. Using data from the Minnesota Longitudinal Study of Risk and Adaptation the same group showed similar effects up to age 32 for academic functioning although, in the case of social functioning, associations with maternal sensitivity were accounted for by confounders such as early socio-economic factors and child's sex (Raby et al., 2014). Compelling though they are, studies such as these cannot rule out genetic confounds. However, van der Voort and colleagues (2014) addressed this possibility in a longitudinal study of children adopted in infancy (parent-child dyads had no biological relation ruling out genetic confounding) and found that maternal sensitivity during infancy predicted internalising symptomatology during adolescence. A causal role for maternal sensitivity in development is further supported by clinical trials of attachment-based intervention programmes that show that rates of insecure or disorganised attachment can be reduced by increasing maternal sensitivity (Juffer, Bakermans-Kranenburg, & van IJzendoorn, 2005; Van IJzendoorn, Juffer, & Duyvesteyn, 1995).

#### **8.1.2.1 Sensitivity to distress and to non-distress**

Methods to assess maternal sensitivity vary considerably in the extent to which they use home or lab based observations, whether the conditions are standardised, the duration of the observations, and their coding. It may be that these broad characterisations ignore possible issues of domain specificity whereby aspects of sensitivity that entail different processes may have different developmental consequences (Grusec & Davidov, 2010). In particular, maternal sensitivity to infant bids for reciprocity in playful interactions are likely to promote joint exploration and joint attention (Hobson, Patrick, Crandell, Perez, & Lee, 2004) and hence cognitive development (Bornstein & Tamis-Lemonda, 1997) but they do not appear to contribute to attachment security (Murray et al., 2008). In contrast, sensitive comforting responses to infant distress are associated with attachment security (Leerkes, 2011) but not cognitive development (McElwain & Booth-Laforce, 2006). Moreover, it has been suggested that sensitivity to distress and sensitivity to non-distress may have different antecedents, with maternal sensitivity to non-distress being associated with socio-demographic risk (e.g. age, education, income, uninvolved partner) and sensitivity to distress being associated with the caregiver's emotional and cognitive competencies and responses to the infant's negative emotions (Leerkes, Crockenberg, & Burrous, 2004; Leerkes, Weaver, & O'Brien, 2012; Leerkes, 2010).

#### **8.1.3 Current study**

In the current study, we investigated if CD measured during pregnancy in primiparous mothers was a significant predictor of maternal insensitivity during mother-infant interaction at 29 weeks. We predicted that high level of CD at 32 weeks gestation would

be associated with maternal insensitivity during early mother-infant dyadic communication controlling for plausible confounders. Because, both sensitivity to distress and non-distress may have both distinct antecedents in mothers and different consequences to the infant's socio-cognitive development, we examined the contribution of CD to each.

## **8.2 Methods**

### **8.2.1 Design**

The current study draws on data from the Wirral Child Health and Development Study (WCHADS; Sharp et al., 2012). The WCHADS is a prospective longitudinal study that aims to identify early social, emotional and biological risks involved in the development of childhood conduct problems.

First-time mothers were recruited consecutively from an antenatal clinic to establish a general population ('extensive') sample, from which a ('intensive') subsample stratified by psychosocial risk (partner psychological abuse), was drawn, and both were then followed in tandem. This two stage stratified design enables intensive measurement, in a subsample such as those used in this study to assess CD and maternal sensitivity, while collection of other measures across the whole 'extensive' sample allow a weighting back of the findings from the intensive subsample to give general population estimates. A detailed flowchart of the sampling and recruitment procedure can be found elsewhere (Sharp et al., 2012).

The extensive sample comprised primiparous mothers ( $\geq 18$  years of age and English speaking) who sought antenatal care at 12 weeks gestation between February

2007 and October 2008 at the Wirral University Teaching Hospital. Demographic information was collected at this time.

At 32 weeks, Mothers in the intensive sample completed Five Minute Speech Samples (FMSS; Leeb et al., 1991). The task was part of a comprehensive set of interviews and assessments mothers were asked to complete. The speech samples were audio-recorded and later transcribed by members of the WCHADS team. At 29 weeks into the post-natal period, mothers completed a 15-min play protocol with their babies in the research base, from which maternal sensitivity was coded (The NICHD Early Child Care Research Network, 1999). Approval for the procedures, Reference Number 05/Q1506/107 was obtained from the Cheshire North and West Research Ethics Committee (UK).

### **8.2.2 Participants**

From a total of 2158 women initially approached, 68.4% (1286) consented to taking part in the study and completed pre-natal screening (20 weeks). A stratified random subsample of 316 mothers was recruited to the intensive sample at 32 weeks gestation on the basis of their prior responses to a measure of partner psychological abuse. Five-minute speech samples (FMSS) were available for 287 women and 272 mother-infant dyads were later observed in interaction at 29 weeks of age as part of a comprehensive assessment from which maternal sensitivity scores were derived. Since only a subgroup of interactions included some naturally occurring infant distress complete speech sample and postnatal interaction data was available from 237 dyads for sensitivity to non-distress and from 180 dyads for sensitivity to distress, respectively.



### 8.2.3 Measures and procedure

#### 8.2.3.1 Communication Deviance (CD)

The CD coding system was originally developed for family interaction (Velligan, 1985). The system captures eight different types of communication disturbances that were based on previous work on CD (Doane & Singer, 1977; Singer & Wynne, 1965a, 1965b, 1966; Wynne et al., 1977; Wynne & Singer, 1963a, 1963b), namely: (1) *abandoned, abruptly ceased, uncorrected remarks*; (2) *unintelligible remarks*; (3) *contradictions, denials and retractions*; (4) *ambiguous referents*; (5) *extraneous questions and remarks*, (6) *tangential, inappropriate responses to questions or remarks*; (7) *odd word usage/odd sentence construction*; and (8) *reiteration*. Table 1 shows definitions and examples of the different codes.

CD scores were calculated as the number of instances of CD divided by the number of words spoken to account for verbosity (as suggested in the literature *see* Hirsch & Leff, 1971; Miklowitz & Stackman, 1992). This coding protocol has been shown to have good reliability and construct validity against other methods of assessing CD (Velligan, Goldstein, Nuechterlein, Miklowitz, & Ranlett, 1990) and has been previously used with clinical (Velligan et al., 1996; Velligan, Funderburg, Giesecke, & Alexander, 1995) and high-risk populations (Velligan et al., 1988). The system has also been previously applied to natural speech samples (Docherty, 1993) and to five-minute speech samples (Kymalainen, 2005; Kymalainen, Weisman, Rosales, & Armesto, 2006).

<i>CD code</i>	<i>Definition</i>	<i>Example</i>
Abandoned, abruptly ceased, uncorrected remarks	Speaker abruptly abandons an idea without returning to it leaving a sense of no closure.	<i>“M: You know, what does it...I wanna look like that you know. So it wasn’t...That’s, I think that’s what was sort of so err, hard.”</i>
Unintelligible remarks	Speaker makes remarks that are not understandable in the context of conversation.	<i>“M: At the moment I feel like... ‘cause even, we had a doctors appointment yesterday morning and we still can’t categorically say we know a lot about genetically what happens, what the baby’s made of so I don’t think many people know that you see.”</i>
Contradictions, denials and retractions	Speaker contradicts, openly retracts or denies what he has previously said.	<i>“M: That’s all really, I’m just happy about it (...) M: I <u>don’t</u> know how I feel.”</i>
Ambiguous referents	Speaker uses linguistic referents that are unclear or ambiguous and that could be referring to more than one person or object.	<i>“M: I maybe don’t allow myself as much of <u>that</u> as what maybe I should do because I’m always focussed on making sure everything’s okay, you know.”</i>
Extraneous questions and remarks	Speaker makes comments or asks questions that are extraneous to the task.	<i>“M: What do people normally say? M: It’s very strange being asked to ramble”</i>

Tangential, inappropriate responses to questions or remarks	Speaker makes non-sequitur replies to questions or remarks.	<i>“(...) Err, chest of drawers and we just need to get a little wardrobe and I’ve got like this lamp, a Winnie the pooh lamp, that plays music and stuff and you can get like a Winnie the Pooh thing to put over the cot and stuff, make it all dead nice. It doesn’t have to be Winnie the Pooh but I thought Winnie the Pooh would be nice, plus [partner’s name]’s mum gave us some Winnie the Pooh pictures for the walls so that’s made us decide Winnie the Pooh.”</i>
Odd word usage/odd sentence construction	Speaker uses of words or sentences in a way that is odd, incorrect or out of context.	<i>“M: I feel like quite protective over her even though she’s not here <u>already</u>.”</i>
Reiteration	Speaker repeats the same thought, idea or word several times without adding new information.	<i>“M: I think I <u>probably</u> worry <u>probably</u> as a tendency more than <u>probably</u> most people would but then that’s <u>probably</u> because I <u>probably</u> am aware of every eventuality.”</i>

**Table 19** - Definitions and examples of the CD codes (Velligan, 1985).

A total of 287 Five Minute Speech Samples (FMSS; Leeb et al., 1991) were coded from the mothers in the 'intensive' sample. In this task the mother was invited to talk for five minutes about her future child using the following prompt:

"I would like to hear your thoughts and feelings about your baby at the moment, in your own words without me interrupting. When I ask you to begin I would like you to speak for 5 minutes, tell me what your impressions have been of your baby whilst you've been pregnant. Have you got any questions before we start?"

Two raters who were blind to all other study variables carried out the coding. For purposes of training, first (P.S.) and second author (K.F.) coded 31% (90) of the speech samples. The training period was preceded by the careful reading of relevant papers in the field of CD (Singer & Wynne, 1966) and the coding manual that was kindly provided to us by its author (Velligan, 1985). Both first and second author were blind to any background information about the study's participants and were only provided with anonymised transcripts and audio-recordings (the only information available was the participants id number). Frequent meetings were held at the University of Liverpool. Following a period of training, both coders independently scored a subset of speech samples representing ~10% (30) of the total sample. Some of the CD codes were very infrequent (e.g. reiteration) but the estimated reliability was good (intraclass correlations for the different items ranged from .77 to .97).

### **8.2.3.2 Maternal sensitivity at 29 weeks**

Maternal sensitivity was assessed with a 15-min standard laboratory-based protocol (NICHD Early Child Care Research Network, 1999). Mothers were asked to play with their infants seated in a reclining chair or on the floor mat as they would at home. During the free-play, mothers were asked to play with their children using the following prompt:

“Play as you might usually do with your baby”

The total 15 minutes of play were video recorded. During the initial seven minutes, mothers were instructed to play with their babies using a toy of their choice. After the initial seven minutes, a researcher knocked on the door and instructed the mother to play for an extra eight minutes with a set of standardised toys provided by the WCHADS team. The camera was placed so that full-face view of the infant and the mother could be captured to enable team to code the dyads eye-to-eye contact.

An investigator from NICHD Early Child Care Research Network trained three raters who were blind to other measures of the study. Subsequently, and blind to the other measures, they coded sensitivity from the video recordings. Each rater achieved good inter-rater reliability for maternal sensitivity on a subset of 30 assessments (intraclass correlations ranged from .85 to .91). These raters scored the video recordings of mother-infant interaction on maternal sensitivity (maternal sensitivity to distress and maternal sensitivity to non-distress) using a 5-point scale, ranging from 1 (not at all characteristic) to 5 (highly characteristic) reflecting mothers’ appropriate, supportive, warm responding to infant communications, playful bids or distress.

### **8.2.3.3 Confounders**

Maternal age and socio-economic deprivation have been found to be important predictors of maternal sensitivity to non-distress (Leerkes et al., 2012) and so were included as potential confounders. Although CD has been found to be unrelated to IQ (e.g. Doane, West, Goldstein, Rodnick, & Jones, 1981), we included a measure of verbal IQ because CD is a measure of verbal communication.

#### **8.2.3.3.1 Index of Multiple Deprivation (IMD)**

Socioeconomic status was determined using the revised IMD (Noble et al., 2004).

According to this system, postcode areas in England are ranked from most deprived (IMD of 1) to least deprived (IMD of 32,482) based on seven domains: (1) income deprivation; (2) employment deprivation; (3) health deprivation and disability; (4) education, skill and training deprivation; (5) barriers to housing and services; (6) living environment deprivation; and, (7) crime. All mothers were ranked according to their area postal code and assigned to a quintile based on the UK distribution of deprivation.

#### **8.2.3.3.2 Verbal IQ**

Verbal IQ was measured with Wechsler Test of Adult Reading (WTAR). The WTAR is a neuropsychological test that takes approximately 10 minutes to complete and that assesses pre-morbid intelligence through the use of fifty irregularly spelled words. During the test, the examiner presents a series of cards with the words prompting the participant for a single pronunciation of the word. The test is stopped when the participant gives 12

consecutive incorrect pronunciations. Each correct pronunciation is given a score of 1 with the maximum raw score of 50. The raw score is then standardised by age and education using published guidelines (Holdnack, 2001). WTAR scores are strongly correlated with measures of verbal IQ, verbal comprehension and full scale IQ (Strauss, Sherman, & Spreen, 2006).

### **8.2.3 Statistical analysis**

In order to make inference about the general population from our sample, we applied inverse probability weights that accounted for both the stratified sample and sample attrition associated with maternal age, education, depression score at booking, in pregnancy, smoking and marital status. We first examined the simple linear association of CD with overall maternal sensitivity but checked for non-linearity in the association using a lowess regression smooth (Cleveland, 1979) and a “bent-stick” regression that hypothesized that the association was limited to only part of the range of CD scores (Bacon & Watts, 1971). Analyses were repeated for sensitivity in and out of the context of infant distress, with different weights to account for the fact that a substantial proportion of the infants did not become distressed during the observation. All analyses were carried out in Stata 13, the bent-stick regression using a procedure written by Mark Lunt.

	Extensive sample n= 974	Intensive sample n= 315	
Age			
Mean (SD)	26.71 (5.79)	27.57 (6.08)	$t(1283) = -2.28, p = .023$
Missing	4 (0.4%)	4 (0.3%)	
Sex (baby)			
Male	N/a	159 (50.5%)	
Female	N/a	156 (49.2%)	
Missing	N/a	1 (0.3%)	
Marital status			
Single	113 (11.6%)	37 (11.7%)	$\chi^2(6) = 3.83, p = .7$
Married	373 (38.3%)	127 (40.3%)	
Divorced	1 (0.1%)	1 (0.3%)	
Separated	2 (0.2%)	2 (0.6%)	
Cohabiting	367 (37.7%)	106 (33.7%)	
Partner lives elsewhere	110 (11.3%)	40 (12.7%)	
Other	3 (0.3%)	1 (0.3%)	
Missing	5 (0.5%)	1 (0.3%)	



Verbal IQ			
	N/a	105.45 (6.77)	
Education			
No qualifications	106 (10.9%)	18 (5.7%)	$\chi^2(10) = 16.76, p = .08$
CSE	15 (1.5%)	4 (1.3%)	
NVQ	302 (31%)	99 (31.4%)	
GNVQ	60 (6.2%)	17 (5.4%)	
A levels	270 (27.7%)	114 (36.2%)	
HNC	3 (0.3%)	1 (0.3%)	
HND	14 (1.4%)	1 (0.3%)	
Further qualifications	7 (0.7%)	2 (0.6%)	
Undergraduate degree	28 (2.9%)	14 (4.4%)	
Postgraduate degree	5 (0.5%)	1 (0.3%)	
Other	113 (11.6%)	38 (12.1%)	
Missing	51 (5.2%)	6 (1.9%)	
Ethnicity			
White British	934 (95.9%)	302 (95.9%)	$\chi^2(1) = 15.55, p = .113$
Other	35 (3.7%)	13 (4.1%)	
Missing	5 (0.5%)	0 (0.0%)	
Index of multiple deprivation (IMD)			
Score	33.03 (20.22)	32.1 (20.07)	$t(1281) = .716, p = .474$

Rank (1 = most deprived)		
1	413 (42.4%)	119 (37.8%)
2	174 (17.9%)	62 (19.7%)
3	223 (22.9%)	85 (27%)
4	88 (9%)	21 (6.7%)
5	70 (7.2%)	28 (8.9%)
Missing	6 (0.6%)	0 (0.0%)

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**Table 20** - Frequencies and descriptives of the demographic variables in the study (unweighted).

Assessment period	Variable	N	Mean (s.d.)	Min	Max
20 weeks gestation	Maternal age	237	26.96 (5.96)	0	47
	Verbal IQ	237	105.68 (6.43)	88	118
	IMD (quintiles)	237	2.29 (1.3)	1	5
32 weeks gestation	CD <sup>1</sup>	237	0 (1)	1	-1.2
29 weeks postnatal	Overall sensitivity <sup>1</sup>	237	0 (1)	-2.67	1.42
	Sensitivity to non-distress <sup>1</sup>	237	0 (1)	-2.7	1.36
	Sensitivity to distress <sup>1</sup>	180 <sup>2</sup>	0 (1)	-2.26	1.41

**Note:** <sup>1</sup>Sensitivity and communication measures have been standardised.

<sup>2</sup>not all infants became distressed so sensitivity to distress is available for only a subset of mothers.

**Table 21** - Means, standard deviations and ranges (weighted).

## 8.3 Results

Table 20 shows the means, standard deviations and frequencies for the demographic variables for both the intensive and extensive sample (unweighted) and table 21 the

Overall maternal sensitivity (n= 237)				
	B (S.E.)	95% CI		Model Summary
<b>CD</b>	-.385 (.092) ***	-.567	-.203	<i>F</i> (1,236)= 17.38 *** <i>R</i> <sup>2</sup> = .078
Constant	.349 (.103) ***	.146	.553	

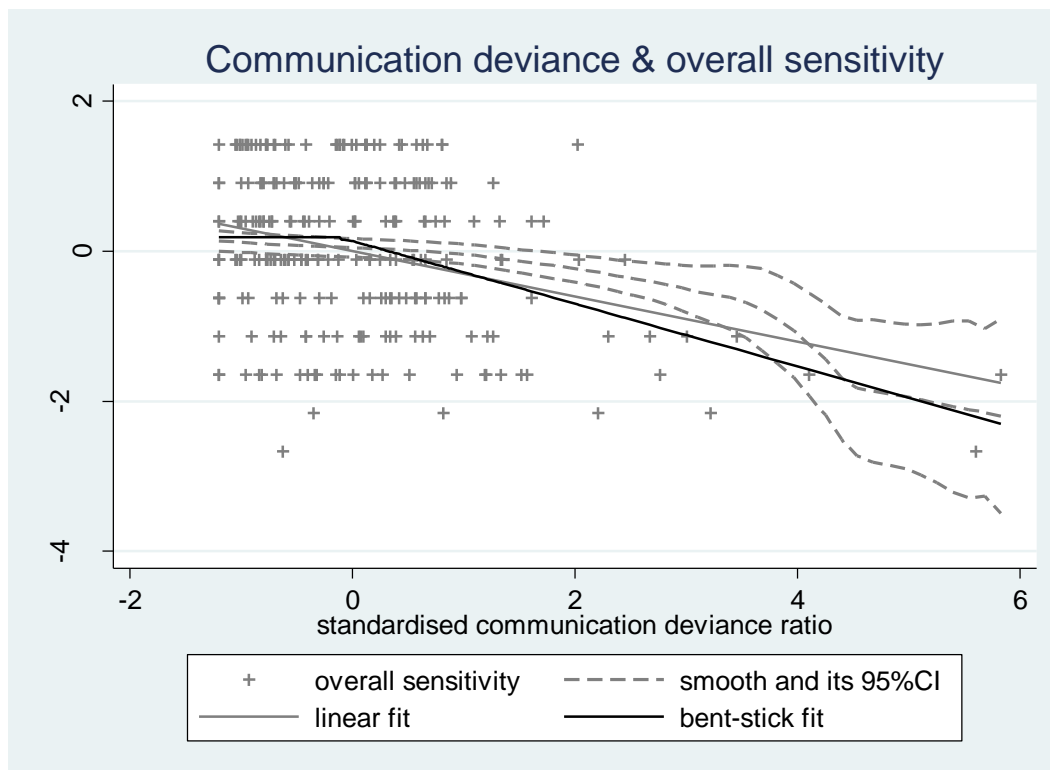
“Bent-stick” regression (n=237)				
	B (S.E.)	95% CI		Model Summary
Breakpoint	-.042 (.148)	-.333	.250	<i>F</i> (2,234)= 12.84 *** <i>R</i> <sup>2</sup> = .099
slope r	-.418 (.106) ***	-.627	-.209	
Constant	.189 (.086) *	.019	.359	

Note: <sup>\*</sup>  $p < .05$  <sup>\*\*</sup>  $p < .01$  <sup>\*\*\*</sup>  $p < .001$

**Table 22** – Weighted bivariate regressions with CD as a predictor of overall maternal sensitivity and summary of the “bent-stick” regression model.

means, standard deviations and ranges for our key variables (weighted) for the intensive sample.

A simple bivariate regression showed a highly significant association with overall sensitivity suggesting that a 1 SD increase in CD was associated with a 0.38 SD decrease in maternal sensitivity (Table 22). However, both variables, in particular CD were skewed.



**Figure 14** – Regression model with lowess smooth, linear and “bent-stick” fit.

Figure 14 shows the fitted regression model together with a non-linear regression (lowess smooth). The lowess suggested that the association might be restricted to the upper-end of the distribution of CD scores. A “bent-stick” regression that allowed for the

Overall maternal sensitivity (n= 237)				
	B (S.E.)	95% CI		Model Summary
CD	-.299 (.106)**	-.508	-.091	$F(3,234) = 21.86^{***}$ $R^2 = .243$
Age	.046 (.011)***	.025	.067	
IQ	.032 (.011)**	.011	.053	
Constant	-4.32 (1.12)***	-6.53	-2.11	

Overall maternal sensitivity (n= 237)				
	B (S.E.)	95% CI		Model Summary
CD	-.216 (.076)**	-.365	-.067	$F(4,233) = 19.30^{***}$ $R^2 = .266$
Age	.041 (.010)***	.021	.061	
IQ	.027 (.011)*	.006	.048	
IMD	.123 (.050)*	.024	.221	
Constant	-4.26 (1.07)***	-6.38	-2.15	

Note: \*  $p < .05$  \*\*  $p < .01$  \*\*\*  $p < .001$

**Table 23** - Weighted linear regression with CD as a predictor of overall maternal sensitivity after adjusting for maternal age and verbal IQ and index of multiple deprivation.

lower end of the distribution of CD scores to be of no effect was estimated and is also shown in Figure 14. This suggested that the point of inflection in the regression, though appearing quite close to the lower end of the range of scores occurred fell, because of the skew of the distribution, close to the middle, at the 48<sup>th</sup> percentile. The 95% confidence interval spanned from 37% to 60%. A formal test of the superiority of this model in our stratified sample was not straightforward.

It was possible that the association may have been due to the confounding effects of mother's age or verbal IQ. The regression including these possible confounder showed maternal sensitivity increased strongly with both these variables ( $p < .001$  &  $p = .003$  respectively) and gave a slightly smaller estimated coefficient of -0.30 (95%CI [-.51; -.09]) that was still strongly significant ( $t$ -test  $p = .005$ ). The inclusion of neighbourhood deprivation as an additional confounder reduced the strength of the association further but not its significance.

When sensitivity in and out of the context of infant distress were analysed separately, a significant association was found in both contexts even after accounting for possible the effects of maternal age and verbal IQ. However the effect estimate was substantially larger for the context of distress (-0.42; 95% CI[-0.60; -0.24];  $p < .001$ ) than in non-distress (-0.26; 95% CI[-0.48; -0.03];  $p = .024$ ).

## 8.4 Discussion

The results of the current study indicate that antenatal CD is a significant predictor of maternal sensitivity 29 weeks post-partum. Perhaps more importantly, this seems to be especially the case with maternal sensitivity to distress. These findings should be

Maternal sensitivity to distress (n= 180)				
	B (S.E.)	95% CI		Model Summary
CD	-.418 (.093) ***	-.600	-.235	$F(3,177) = 10.84^{***}$ $R^2 = .177$
Age	.024 (.015)	-.004	.053	
IQ	.019 (.013)	-.006	.045	
Constant	-2.34 (1.31)	-4.92	.237	

Maternal sensitivity to distress (n= 180)				
	B (S.E.)	95% CI		Model Summary
CD	-.293 (.065) ***	-.421	-.164	$F(4,176) = 11.36^{***}$ $R^2 = .216$
Age	.016 (.014)	-.012	.044	
IQ	.014 (.012)	-.009	.039	
IMD	.164 (.059) **	.047	.281	
Constant	-2.34 (1.22)	-4.75	.079	

Maternal sensitivity to non-distress (n= 237)				
	B (S.E.)	95% CI		Model Summary
CD	-.256 (.113) *	-.478	-.034	$F(3,234) = 20.91^{***}$ $R^2 = .229$
Age	.044 (.011) ***	.024	.065	
IQ	.033 (.011) **	.013	.055	
Constant	-4.54 (1.12) ***	-6.74	-2.33	



Maternal sensitivity to non-distress (n= 237)				
	B (S.E.)	95% CI		Model Summary
CD	-.185 (.082)*	-.346	-.024	$F(4,233) = 17.65^{***}$ $R^2 = .247$
Age	.040 (.010)**	.019	.061	
IQ	.030 (.011)**	.009	.051	
IMD	.106 (.050)*	.007	.205	
Constant	-4.48 (1.09)**	-6.62	-2.34	

Note: \*  $p < .05$  \*\*  $p < .01$  \*\*\*  $p < .001$

**Table 24** - Weighted linear regression with CD as a predictor of maternal sensitivity to distress and non-distress after adjusting for maternal age and verbal IQ and index of multiple deprivation.

interpreted in the context of previous research that has reported that maternal sensitivity to non-distress seems to be much more closely predicted by socio-demographic risk factors (Leerkes et al., 2012) whereas sensitivity to distress seems to be much more related to the emotional and cognitive skills and competencies of the mother (e.g. fewer negative emotions in response to infant crying or better skills at detecting infant distress; Leerkes, 2010). These findings should also be interpreted in the larger context of studies that have reported an association between mothers' disrupted communication during face-to-face interaction with their infants and difficulties sensitively attuning to their 4-months-old distress cues (Crockett, Holmes, Granger, & Lyons-Ruth, 2013) as well as initiating and sustaining joint attention bids with the infant (Annie Yoon, Kelso, Lock, & Lyons-Ruth, 2014; Schechter et al., 2010). Moreover, the findings are relevant in the context of the robust association between maternal disrupted communication at 12 to 18 months and children's disorganised attachment style (Madigan et al., 2006). In these studies, disrupted communication is conceptualized as the caregiver's failure to grasp and respond to the intentions conveyed in the infant's communication and it is therefore possible that the communicational problems identified in mothers in our study may reflect specific difficulties with cognitive and emotional processes (e.g. accurate identification of negative emotions and emotional responses to distress, Leerkes & Crockenberg, 2006) which are important in attuning and responding to infant's distress. However, this does not explain the relationship between CD and maternal sensitivity to distress.

One interpretation of these results is to consider that both maternal CD and maternal sensitivity may reflect underlying difficulties with mentalization in the mother. For example, it has been argued that mentalising is likely to be important for repairing misunderstandings during conversation (e.g. using deictic references that the listener finds ambiguous or vague and that lead to misalignment and communication breakdown) and that mentalising and alignment, although dissociable processes, both contribute to successful communication (Pickering & Garrod, 2004, 2006). Mentalising or maternal mind-mindedness have also been found to be a significant and important predictors of children's socio-cognitive development (Meins et al., 2002; Meins et al., 2003). It is therefore, conceivable that the both maternal sensitivity to infant's distress and mother's communication may rely on the same mentalising processes and mechanisms.

Importantly, in the context of the substantial literature on CD in schizophrenia, the association observed between CD and low maternal sensitivity to distress may help to explain previously observed associations between CD and poor social and emotional outcomes in children (Wichstrøm et al., 1996; Wichstrøm, Holte, Husby, et al., 1993; Wichstrøm, Holte, & Wynne, 1993; Wichstrøm et al., 1994) and psychopathology in adult offspring (de Sousa et al., 2013; Roisko et al., 2014; Wahlberg et al., 1997, 2000). Moreover, such disruptions could partly explain the cognitive and affective atypicalities often observed in young children who are later diagnosed with schizophrenia (Welham, Isohanni, Jones, & McGrath, 2009), especially in birth cohort studies (Cannon et al., 2002; Peter Jones, Rodgers, Murray, & Marmot, 1994).

Our findings suggest as well that the impact of CD on child's socio-cognitive development may not be solely mediated by verbal communication and that CD may

have a cross-modal expression, as has been suggested by some previous investigators (Lewis, Rodnick, & Goldstein, 1981). Hence, the detrimental effect of CD may be observable in early development as suggested previously (Velligan et al., 1988), and at a more basic level of maternal relatedness with the preverbal infant (Wynne, 1988).

An important strength of the present study was that CD was measured before the birth of the child, eliminating the possibility of evocative effects of infant-related variables on the parent as raised previously in the CD literature (Miklowitz & Stackman, 1992). However, the study also has several limitations, which should be noted. First, because of its goals (to eliminate the possibility of evocative effects of the infant on the parent) communication was not measured when both mother and infant were interacting. Also, our five-minute speech samples are not an everyday conversation; they reflect soliloquies rather than dialogues and it could be argue that CD scores were inflated by the experimental condition. In the future, it will be informative and relevant to measure CD at multiple time-points (including before birth) during the infant's development and test, not just its stability across time, but also across setting (e.g. when mother-infant are interacting). Also, it would be relevant to include other developmental constructs (e.g. joint attention) as well as infant-related variables (e.g. theory of mind, attachment, etc.).

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## **Chapter 9. Conclusion**

## 9.1 Conclusions and future research

The investigations in this thesis cover different empirical questions on both the social predictors and the psychological and affective processes involved in TD. Each chapter contains its own summary of the findings, conclusions and considerations for future research and therefore the present section will be focused on an overall conclusion of the investigations with the aim to explore ideas for future research.

The work started with the review presented in **Chapter 2**. As mentioned, several psychological processes and theories have been developed across the years to explain TD and related constructs. However, very few studies have attempted to test the interplay between these cognitive mechanisms and affect during conversation. This is especially relevant first because TD is expressed in communication, and second because of the impact of emotional-salience on TD (i.e. affective reactivity of speech), which stands as the most well-replicated phenomenon in the field of both communication disturbances (e.g. Docherty, 1996) and TD (e.g. Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995).

The study presented in **Chapter 3** tried to address this gap in the literature through the use of a repeated-measures design to test both the role of negative affect and internal source monitoring in TD (within and between subjects). The affective reactivity of speech effect was replicated in patients diagnosed with schizophrenia and also, although to a lesser degree, in healthy volunteers. It was also reported robust associations between poorer performance on the internal source-monitoring task and TD. More importantly, discrimination scores mediated the relationship between negative affect and

TD in both the neutral and the salient interviews, separately. Although, the meditational analysis using change scores showed that the calculated difference in negative affect across the interviews did not predict either the change in the discrimination scores or the change in TD scores across the interviews (however, in this analysis change in discrimination scores was a significant and robust predictor of change in TD scores) this may have been because of the additional noise introduced by using scores of this kind.

These results provide partial support to the more overarching hypothesis that the jumbled up quality of TD (e.g. incoherence) is explained by either the inadvertent verbalisation of inner speech or the omission of segments of speech due to the temporary worsening of the individual's ability to source monitor self-generated cognitions whilst experiencing negative affect or emotional arousal.

Of note is that the clinical group already displayed more TD and a poorer discrimination scores in the neutral interview than the comparisons, suggesting that other variables may also account for these differences. Moreover, the internal source monitoring mechanism is unlikely to explain instances of negative TD (e.g. poverty of speech, see **Chapter 2** section 2.7.2).

In **Chapter 4**, a more exploratory set of hypotheses was addressed, in which the focus was on trying to understand the role of both inner speech and self-concept in TD. This study was stimulated by dialogical self theory (Hermans, Kempen, & Van Loon, 1992) and by its implications for the understanding of psychotic experiences (Lysaker & Lysaker, 2006). Of especial interest were the hypotheses that negative TD may reflect a suspension of inner dialogue and that positive TD may reflect poorer dialogical coherence. The analyses revealed that the negative dimension of TD (poverty of speech

and tangentiality) was robustly associated with less self-reported inner speech (e.g. dialogical, and evaluative/motivational inner speech) and that the disorganised dimension of TD (e.g. derailment, incoherence, illogicality, etc.) was significantly correlated with less clarity of self-concept (it is important to note that these analyses controlled for co-morbid psychotic experiences such as hallucinations and delusions).

One of the interesting implications of these findings is that they provide evidence that the negative dimension of TD may reflect an overall impoverishment of verbal thought and that this dimension may be better described as poverty of inner speech instead of poverty of speech. This finding also has implications for the debate around whether TD is a communicational, speech or cognitive disturbance (see Chaika, 1982; Lanin-Kettering & Harrow, 1985). The findings seem to highlight the limitations of using solely a linguistic approach to study TD (see **Chapter 2**). In addition, it would be relevant to test social predictors and environmental factors associated with this impoverishment of verbal thought, especially given that this is a prominent and enduring feature in older patients diagnosed with schizophrenia (e.g. Bowie et al., 2005).

In **Chapter 5**, the investigation shifted to the problem of understanding the social predictors of TD. To discover that no work had previously been carried out testing the role of social isolation in TD was surprising (**Chapter 2**) for two reasons: first, social isolation has been implicated in the maintenance and development of other psychotic experiences such as hallucinations and delusions (e.g. Hoffman, 2007); and second, as mentioned before, TD is expressed in communication and conversation which are social activities.

This study reported robust and significant association between increased social isolation (reflecting not just smaller social networks but less social contact within these networks) and TD. These analyses were especially relevant because they controlled for comorbid psychotic experiences such as presence of hallucinations and delusions, suggesting specificity. Obviously, the study does not clarify the direction of causality; it may well be that the presence of TD may lead to social distance and consequently to social isolation. However, if this were to be the case, then one would expect severity of other psychotic symptoms to be equally associated with increased social isolation and they were not.

In future studies, it would be interesting to understand how social isolation may impact on both source monitoring ability (**Chapter 3**), impoverishment of inner speech (**Chapter 4**) or the socio-cognitive mechanisms that have been reported to be associated with TD (**Chapter 2**). Moreover, social isolation may well be a maintaining factor for TD, which in turn could lead to the development of new interventions. As mentioned before in **Chapter 2** only a handful of therapeutic strategies have actually been tested for TD (e. g. video-taped feedback, Satel & Sledge, 1989). In this context, it would be interesting to test the effectiveness of interventions that target a social isolation (providing communicational opportunities to patients) in ameliorating TD. As mentioned in **Chapter 2**, with the exception of a couple of studies (e.g. Grant & Beck, 2009) the role of interpersonal environment in TD has hardly received any attention.

**Chapter 6** presents a comprehensive review of the literature on CD. The interest in this familial variable stems from both its historical value and its specific association with TD (e.g. Sass, Gunderson, Singer, & Wynne, 1984; Singer & Wynne, 1965;



Wahlberg et al., 1997). The review assessed the balance of the evidence with regards to the relationship between CD and TD, and tried to address the more prominent methodological and conceptual issues in this area of research. Overall, there was some evidence to suggest that parental CD is a significant predictor of TD in the offspring and plausible developmental pathways and mechanisms to explain this relationship were advanced. Of note is that this literature has important limitations, for example, no study to date has tested the specificity of the relationship between CD and TD by controlling for comorbid psychotic experiences in the offspring. It would be relevant in future studies to investigate these relationships more carefully by measuring other psychotic experiences and controlling statistically for their occurrence. Also, given that TD is a transdiagnostic construct, it would be relevant to use multi-diagnostic samples.

In **Chapter 7** work on CD was continued by means of a meta-analysis. There was a robust overall effect-size for the studies that have tested CD in parents of psychotic offspring ( $g = .97$ ). Perhaps, more importantly, when the data was sub-analysed by parental groups there were significant between-group differences with a much more robust effect-size for mothers ( $g = .89$ ) than fathers ( $g = .39$ ). Unfortunately, there were not enough data to test the association between parental CD and TD.

The results should be interpreted in the context of the larger review of the field presented in the previous chapter (**Chapter 6**) where specific ways in which the field can be moved forward are discussed (along with potential mechanisms of action and developmental pathways). It is suggested that Maternal CD may act as a distal risk factor for TD though its potential role via episodes of joint attention, given that CD is conceptualised as a difficulty establishing and maintain a focus of attention.

There is some evidence from birth cohort studies to suggest that difficulties at the level of caregiver-infant relationship are associated with increase risk of psychosis in the offspring (e.g. Cannon et al., 2002; Jones, Rodgers, Murray, & Marmot, 1994) and several studies that have reported socio-cognitive deficits (of relevance to communication) in parents of patients diagnosed with schizophrenia (e.g. *mentalisation*, Lavoie et al., 2013, 2014).

In **Chapter 8**, and with the findings of **Chapter 6** and **7** in mind, a large study with primiparous mothers was conducted using data from an ongoing cohort research programme. The goal was to test if maternal CD was associated with maternal sensitivity given that this latter variable is a strong predictor of infant's cognitive and socio-affective development (e.g. Leerkes, Nayena Blankson, & O'brien, 2009). Of note is that in this study, CD was measured before the birth of the first child at 32 weeks gestation (in an attempt to exclude the evocative effect of the child's behaviour on the mother), and maternal sensitivity was measured at 32 weeks after birth, whilst the mothers played with their infants. There were significant associations between CD and less maternal sensitivity to both infant's distress and non-distress cues (however, the association was much more robust with the former).

One way of taking these findings further would be to test more complex models using CD measured across different time-points and in different contexts (e.g. during mother-child interaction). On this point, it is relevant to note that very few studies have tested the longitudinal stability of CD (see **Chapter 6**). Also, very rarely have researchers used more than one CD methodology. It would be important to measure CD using both family-based interaction schemes (e.g. Velligan, Goldstein, Nuechterlein, Miklowitz, &

Ranlett, 1990) and individual methodologies (e.g. Docherty, Rhinewine, Labhart, & Gordinier, 1998) along with conversational alignment tasks (e.g. request/response task, Stewart, Corcoran, & Drake, 2008) and social cognition tasks (e.g. hinting task, Corcoran, Mercer, & Frith, 1995). It would also be pertinent to explore both the association between caregiver's CD and 'theory-of-mind' (ToM) development in the child and the mediating role of maternal sensitivity and joint attention given that ToM is a cognitive mechanism of interest in TD (see **Chapter 2** section 2.7.3).

There is evidence from developmental studies to suggest that maternal communication about child's mental states is a powerful predictor of the child's socio-cognitive development (e.g. Meins, Fernyhough, Fradley, & Tuckey, 2001). If a link between maternal CD and infant's ToM development was to be found, this would provide the first developmental pathway to explain some of the difficulties involved in TD.

Finally, at the end of **Chapter 2** (section 2.10), a tentative cognitive-developmental model of TD is presented. The model was developed with the aim of fomenting research on TD but also with clinical practice in mind given the recognised need to develop specific social, behavioural and cognitive therapeutic strategies to address TD.

In this model, potential relationships between some of the variables discussed in this thesis are described. An interesting research programme would be to test this model or at least explore some of the proposed relationships in more detail. For example, as we mentioned before, the role of social isolation has rarely been explored in its association with cognitive mechanisms of psychotic experiences. Regarding TD, it would be interesting to replicate the findings presented in **Chapter 5** using more comprehensive

methodologies to assess social isolation (e.g. social network analysis) that allow for the exploration of important variables such as density of the social network (i.e. degree of connectedness or cohesion) and not just size of the network.

It would also be pertinent to understand how social isolation may impact on physiological and biological correlates of TD and not just on cognitive mechanisms such as internal source monitoring or ToM. For example, it has been suggested that thought-disordered patients show an attenuation of event-related potentials' (ERP) component N400 when exposed to context-incongruent linguistic stimuli (e.g. Kuperberg, McGuire, & David, 1998). The attenuation of this negative-going inflection on the EEG is assumed to index *insensitivity* to linguistic context. In line with this, it would be relevant to investigate if social isolation (and family communication) is associated with the attenuation of this ERP component. It is important to note that some researchers have identified specific failures at the level of deictic linguistic references (that rely heavily on the linguistic context) in patients and their relatives (e.g. Docherty, Miller, & Lewis, 1997) whereas others have suggested that TD is associated with specific difficulties in the integration of contextual information (Hardy-Baylé, Sarfati, & Passerieux, 2003). It may well be that such difficulties could be accounted for, at least in part, by the toxic and enduring effects of social isolation over time.

The model also suggests a state-dependent deterioration in the internal source monitoring ability and ToM through the impact of arousal and negative affect elicited by the conversational context. In this regard, the study described in **Chapter 3** had two important limitations. One is related to the fact that ToM was not measured, and the other is related to the non-inclusion of physiological measures of arousal. In a future research

programme, it would be pertinent to test both cognitive mechanisms along with physiological measures of arousal (e.g. electrodermal activity), past history of trauma or adversity and *evaluation sensitivity* (which has been suggested to be a moderator of TD, Grant & Beck, 2009) using a repeated measures design. Such study would provide a better understanding of the role of these different variables in TD.

In this context, it is interesting to note that affective reactivity of speech and TD have been found to be associated with difficulties and fear of social relationships (St-Hilaire & Docherty, 2005) and deactivating attachment strategies in patients (Dozier & Lee, 1995). One possible interpretation is that interpersonal threat triggered by a social environment that is perceived by the patient as threatening and invalidating may be particularly taxing to the patient's already depleted cognitive resources (as suggested by Beck, Rector, Stolar, & Grant, 2009). Alternatively, one could suggest that state-dependent changes in ToM may represent a defensive strategy by reducing the amount of perceived interpersonal threat and fear experienced by the patient. It is relevant to mention that the experimental tasks that promote personal disclosure may be especially challenging for these patients (e.g. Haddock et al., 1995) explaining in part the worsening of TD (i.e. affective reactivity of speech). Concurrently, one would expect that the defensive deactivation of ToM and consequent TD would be inversely correlated with paranoia.

More complex to address seems to be the role of semantic priming in TD. As suggested by some researchers (e.g. Beck, Rector, Stolar, & Grant, 2009), it may well be that the hyperpriming effect suggested by authors such as Spitzer (1997) may in fact reflect an hyperpriming to semantic nodes that are closely related to the patient's own

personal worries and concerns. One potential way to explore this hypothesis would be to use two lexical decision tasks, one that includes primes and targets that are personally significant to the patient along with standardised stimuli and a second more standardised task. Such emotional-salient stimuli could be easily assembled with an inventory of the individual's personal concerns and worries. More importantly, it would provide an explanation for the conflicting findings reported in the semantic priming literature (see **Chapter 2**).

Along with this study, it would be relevant to test the impact of personally significant and salient stimuli in thought-disordered individuals as suggested by Harrow and colleagues (1983) in their seminal study on *intermingling* in TD. One possible way of doing this would be to modify a task such as the Rey Auditory Verbal Learning Test (Rey, 1964) where again some of the word-stimuli would correspond to the individual's personal concerns and worries. Underlying such study is the hypothesis that disorganised forms of TD are associated with the cognitive and affective interference and intrusion of personally salient stimuli. Consequently, one would expect thought-disordered patients to display more interference of personally salient stimuli in this task.

Finally, it is important to note as well that research on TD is likely to be greatly improved with the development of quicker and less laborious methods for its assessment and quantification. In recent years, researchers have attempted to develop self-report questionnaires (e.g. Barrera, McKenna, & Berrios, 2008) and automated methods to quantify TD (e.g. Elvevåg, Foltz, Weinberger, & Goldberg, 2007). Such endeavour, if successful, would allow researchers to explore the relationships between these variables using larger samples.

It is hoped that the work developed in the last 3½ years can help researchers and clinicians who are interested in this field or at least stimulate debate about TD with the final goal of ameliorating and improving the quality of life of service users affected by this symptom.

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## **Appendices**

## Appendix 1. Ethical approval, sponsorship letter and letters of access



### NRES Committee North West - Preston

HRA NRES Centre - Manchester  
Barlow House  
3rd Floor  
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31 January 2013

Professor Richard Bentall  
Professor of Clinical Psychology  
University of Liverpool  
Institute of Psychology Health & Society, Waterhouse Block B, B211, 2nd Floor,  
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L69 3GL

Dear Professor Bentall

**Study Title:** The impact of emotion on cognition and thinking in psychosis  
**REC reference:** 13/NW/0044  
**Protocol number:** UoL000925  
**IRAS project ID:** 118769

The Research Ethics Committee reviewed the above application at the meeting held on 25 January 2013. The Committee thanks Paulo Sousa for attending to discuss the application.

### Documents reviewed

The documents reviewed at the meeting were:

Document	Version	Date
Evidence of insurance or indemnity		02 August 2012
Interview Schedules/Topic Guides	Positive & Negative Syndrome Scale Items	
Interview Schedules/Topic Guides	Salient Interview	
Investigator CV	Richard Bentall	
Investigator CV	Dr William Sellwood	
Investigator CV	Paulo Sousa	
Letter from Sponsor		05 December 2012



Participant Consent Form	Final	08 November 2012
Participant Information Sheet	v3.2	08 November 2012
Protocol	v0.1	08 November 2012
Questionnaire: FTD Scale		
Questionnaire: Psychosis Screening Questionnaire		
Questionnaire: Postive & Negative Affect Schedule		
Questionnaire: Scale for Assessment of Thought, Language and Communication Disorder		
Questionnaire: Source Monitoring Task		
Questionnaire: Thought Control Ability Questionnaire		
Questionnaire: Varieties of Inner Speech Questionnaire		
Questionnaire: Cognitive Intrusions Questionnaire		
Questionnaire: Self-Concept Clarity Scale		
Questionnaire: State-Trait Inventory for Adults (Y-2 Trait items)		
Questionnaire: Lubben Social Network Scale		
Questionnaire: PANSS - Re-ordered		
REC application	3.4	04 January 2013

### Provisional opinion

The Chair welcomed Paulo Sousa to the REC and thanked him for attending to discuss the study. The Committee told Mr Sousa that this is a really good and worthwhile study and that the knowledge gap has been well articulated.

The Committee asked for clarification of the recruitment process and Mr Sousa explained that he will go to see the support workers who will recruit following a favourable ethical opinion, and ask them to consider whether they have any suitable patients.

The Committee asked whether the salient interview is safe or whether it could potentially be damaging. Mr Sousa said that it has been used safely in the past on two occasions. It was done in the context of an inpatient setting so at a sensitive time, but they appreciated the opportunity to talk about these issues.

The Committee asked to see the debriefing sheets which would be used at the end, and Mr Sousa said that he would just be using the information already contained in the Participant Information Sheet for this, and would ensure that participants are aware of numbers to access help out of hours etc.

The Committee asked what would happen if a participant who has become distressed still wants to carry on with the interview, and Mr Sousa said that he will assess capacity at that time. This will be a judgement call and he has experience of assessing in mental health care. If the participant has lost capacity he will not continue with the interview and will not use the information already collected.

Mr Sousa confirmed that he will advertise for healthy volunteers and the Committee asked to see the advert and requested a separate Participant Information Sheet for the healthy volunteers.

The Committee requested changes to the Participant Information Sheet and Consent Form as below

The Committee felt that the salient interview would take longer than 10 minutes given the sensitive open ended questions and asked that this be made clear to participants so they

are aware of the time they are committing. They should also be told that they can edit the recordings if they say something they do not wish to be on record.

The Committee asked that what would happen if the participant disclosed risk of harm be included in the Participant Information Sheet

The Committee asked what would happen if there is an indication of depression, or psychosis in the healthy volunteers, and Mr Sousa stated that the only thing which could be revealed is thought disorder but that if he notices something irregular he will advise them to see their GP. He will also inform them at the debriefing that if any of the questions have caused concern they should contact a relevant party, and will give them relevant contact numbers.

Mr Sousa had no questions for the Committee

The Committee is unable to give an ethical opinion on the basis of the information and documentation received so far. Before confirming its opinion, the Committee requests that you provide the further information set out below.

Authority to consider your response and to confirm the Committee's final opinion has been delegated to the Chair.

**Further information or clarification required**

- a. The Committee would like to see the advert for the healthy volunteers
- b. The Committee would like to see a separate Participant information Sheet for the healthy volunteers
- c. The Committee would like to see the Participant Information Sheets revised to
  - i) State "may discuss" rather than "should" in line 2 of Do I have to take part?
  - ii) Change the first sentence of what will happen if I take part to "I will arrange for us to meet at a mutually convenient time and location"
  - iii) Include "reasonable" before travelling expenses in para 5
  - iv) Clarify the number of questionnaires, how long the salient interview will take (est 30 mins) and that breaks can be built in if necessary
  - v) Include a statement that they can stop the recording at any time and have words deleted or replaced
  - vi) Include the statement "if you disclose information which indicates that you wish to harm yourself or others, confidentiality will have to be broken and I will have to inform the relevant authority"
- d. The Committee would like to see the Consent Form revised to
  - i) Include the sentence "I agree to the use of anonymised direct quotes in the write up of the study"
  - ii) Include the standard clause "I understand that data collected during the study may be looked at by regulatory authorities or by persons from the Trust where it is relevant to my taking part in this study. I give permission for these individuals to have access to this data"

**If you would find it helpful to discuss any of the matters raised above or seek further clarification from a member of the Committee, you are welcome to contact Carol Ebenezer whose contact details are on this letter.**

When submitting your response to the Committee, please send revised documentation where appropriate underlining or otherwise highlighting the changes you have made and giving revised version numbers and dates.

If the committee has asked for clarification or changes to any answers given in the application form, please do not submit a revised copy of the application form; these can be addressed in a covering letter to the REC.

The Committee will confirm the final ethical opinion within a maximum of 60 days from the date of initial receipt of the application, excluding the time taken by you to respond fully to the above points. A response should be submitted by no later than 02 March 2013.

#### **Membership of the Committee**

The members of the Committee who were present at the meeting are listed on the attached sheet.

#### **Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

<b>13/NW/0044</b>	<b>Please quote this number on all correspondence</b>
-------------------	---

Yours sincerely



**Mr Mike Hammond**  
**Vice-Chair**

Email: [nrescommittee.northwest-preston@nhs.net](mailto:nrescommittee.northwest-preston@nhs.net)

*Enclosures: List of names and professions of members who were present at the meeting and those who submitted written comments.*

*Copy to: Mrs Karen Wilding  
Ms Karen Bruce, Merseycare NHS*

**NRES Committee North West - Preston**  
**Attendance at Committee meeting on 25 January 2013**

**Committee Members:**

<i>Name</i>	<i>Profession</i>	<i>Present</i>	<i>Notes</i>
Mr David Abbotts	Lay member	Yes	
Mrs Hannah Chambers	Lay Member	Yes	Co-opted from Liverpool Central
Dr Anoop Chauhan	Consultant Cardiologist	No	
Mr John Dalton	Lay Member	Yes	
Mrs Debbie Foord	Service Improvement Manager	Yes	
Mr Mike Hammond	Lay Member	Yes	
Ms Eleanor Jolley	Lay Member	No	
Mrs Kate Kilshaw	Radiographer	Yes	
Ms Joanna Marshall	Nurse Specialist in Acute Pain	Yes	
Dr Rob Monks	Senior Lecturer Department of Nursing	Yes	
Professor Videsh Raut	Consultant Orthopaedic Surgeon	No	
Dr Valerie E Siddall	Retired Senior Manager - Pharmaceutical Industry	Yes	Co-opted from Haydock
Mrs Valerie Skinner	Nurse (Retired)	Yes	Co-opted from Lancaster
Mrs Vasanthi Vasudevan	Diabetes Research Nurse	No	
Dr Patricia Wilkinson	General Practitioner	No	

**Also in attendance:**

<i>Name</i>	<i>Position (or reason for attending)</i>
Mrs Carol Ebenezer	Committee Co-ordinator

**NRES Committee North West –Preston**

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05 February 2013

Professor Richard Bentall  
Professor of Clinical Psychology  
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Institute of Psychology Health & Society, Waterhouse Block B, B211, 2nd Floor,  
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Dear Professor Bentall

<b>Study title:</b>	<b>The impact of emotion on cognition and thinking in psychosis</b>
<b>REC reference:</b>	<b>13/NW/0044</b>
<b>Protocol number:</b>	<b>UoL000925</b>
<b>IRAS project ID:</b>	<b>118769</b>

Thank you for your email of 01 February 2013, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair.

We plan to publish your research summary wording for the above study on the NRES website, together with your contact details, unless you expressly withhold permission to do so. Publication will be no earlier than three months from the date of this favourable opinion letter. Should you wish to provide a substitute contact point, require further information, or wish to withhold permission to publish, please contact the Co-ordinator Carol Ebenezer, [nrescommittee.northwest-liverpoolcentral@nhs.net](mailto:nrescommittee.northwest-liverpoolcentral@nhs.net).

**Confirmation of ethical opinion**

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

**Ethical review of research sites**

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

### Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

*Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.*

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

*Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.*

*For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.*

*Sponsors are not required to notify the Committee of approvals from host organisations*

**It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).**

### Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Advertisement		
Covering Letter		
Evidence of insurance or indemnity		02 August 2012
Interview Schedules/Topic Guides	Positive & Negative Syndrome Scale Items	
Interview Schedules/Topic Guides	Salient Interview	
Investigator CV	Richard Bentall	
Investigator CV	Dr William Sellwood	
Investigator CV	Paulo Sousa	
Letter from Sponsor		05 December 2012
Participant Consent Form		31 January 2013
Participant Information Sheet: Healthy Volunteers	v3.3	31 January 2013
Participant Information Sheet: Patients	v3.3	31 January 2013
Protocol	v0.1	08 November 2012
Questionnaire: FTD Scale		
Questionnaire: Psychosis Screening Questionnaire		
Questionnaire: Positive & Negative Affect Schedule		
Questionnaire: Scale for Assessment of Thought, Language and Communication Disorder		

Questionnaire: Source Monitoring Task		
Questionnaire: Thought Control Ability Questionnaire		
Questionnaire: Varieties of Inner Speech Questionnaire		
Questionnaire: Cognitive Intrusions Questionnaire		
Questionnaire: Self-Concept Clarity Scale		
Questionnaire: State-Trait Inventory for Adults (Y-2 Trait items)		
Questionnaire: Lubben Social Network Scale		
Questionnaire: PANSS - Re-ordered		
REC application	3.4	04 January 2013
Response to Request for Further Information		

### **Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

### **After ethical review**

#### **Reporting requirements**

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

#### **Feedback**

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

<b>13/NW/0044</b>	<b>Please quote this number on all correspondence</b>
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We are pleased to welcome researchers and R & D staff at our NRES committee members' training days – see details at <http://www.hra.nhs.uk/hra-training/>

With the Committee's best wishes for the success of this project.

Yours sincerely

A handwritten signature in black ink, appearing to read 'P Wilkinson', with a stylized flourish at the end.

**Dr Patricia Wilkinson**  
**Chair**

Email: [nrescommittee.northwest-liverpoolcentral@nhs.net](mailto:nrescommittee.northwest-liverpoolcentral@nhs.net)

*Enclosures:* "After ethical review – guidance for researchers"

*Copy to:* Mrs Karen Wilding  
Ms Karen Bruce, MerseyCare NHS



**NRES Committee North West - Preston**

HRA NRES Centre - Manchester  
Barlow House  
3rd Floor  
4 Minshull Street  
Manchester  
M1 3DZ

Tel: 0161 625 7818  
Fax: 0161 625 7299

06 December 2013

Professor Richard Bentall  
Professor of Clinical Psychology  
University of Liverpool  
Institute of Psychology Health & Society, Waterhouse Block B, B211, 2nd Floor,  
University of Liverpool, 1-5 Brownlow Street  
Liverpool  
L69 3GL

Dear Professor Bentall

<b>Study title :</b>	<b>The impact of emotion on cognition and thinking in psychosis</b>
<b>REC reference:</b>	<b>13/NW/0044</b>
<b>Protocol number:</b>	<b>UoL000925</b>
<b>Amendment number:</b>	<b>1</b>
<b>Amendment date:</b>	<b>18 November 2013</b>
<b>IRAS project ID:</b>	<b>118769</b>

Increase in sample size

The above amendment was reviewed by the Sub-Committee in correspondence.

**Ethical opinion**

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

### Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Notice of Substantial Amendment (non-CTIMPs)	1	18 November 2013

### Membership of the Committee

The members of the Committee who took part in the review are listed on the attached sheet.

### R&D approval

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

### Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

We are pleased to welcome researchers and R & D staff at our NRES committee members' training days – see details at <http://www.hra.nhs.uk/hra-training/>

<b>13/NW/0044:</b>	<b>Please quote this number on all correspondence</b>
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Yours sincerely



**Dr Patricia Wilkinson**  
**Chair**

E-mail: [nrescommittee.northwest-liverpoolcentral@nhs.net](mailto:nrescommittee.northwest-liverpoolcentral@nhs.net)

*Enclosures:*                      *List of names and professions of members who took part in the review*

*Copy to:*                          *Ms Karen Bruce, MerseyCare NHS Trust*  
   *Mrs Karen Wilding*

**NRES Committee North West - Preston**

**Attendance at Sub-Committee of the REC meeting on 06 December 2013**

<i>Name</i>	<i>Profession</i>	<i>Capacity</i>
Mr Ken Cook	Acute Care Manager (retired)	Expert
Dr Patricia Wilkinson	General Practitioner/ Chair	Expert

**Also in attendance:**

<i>Name</i>	<i>Position (or reason for attending)</i>
Mrs Carol Ebenezer	REC Manager



**Prof Richard Bentall**  
Institute of Psychology, Health  
and Society  
Block B Waterhouse Building  
1-5 Brownlow Street  
Liverpool  
L69 3GL

**Mrs Lindsay Carter**  
**Clinical Research Governance**  
**Manager**

University of Liverpool  
Research Support Office  
2nd Floor Block D Waterhouse  
Building  
3 Brownlow Street  
Liverpool  
L69 3GL

05 December 2012

Tel: 0151 794 8722

Sponsor Ref: UoL000925

Email: [lcarter@liverpool.ac.uk](mailto:lcarter@liverpool.ac.uk)

**Re: Sponsorship Approval**

***"The Impact of emotion on Cognition and Thinking in Psychosis"***

Dear Prof Richard Bentall

After consideration at the Sponsorship Review and Approval Committee on 4<sup>th</sup> December 2012 I am pleased to confirm that the University is prepared to act as Sponsor under the Department of Health's Research Governance Framework for Health and Social Care 2<sup>nd</sup> Edition (2005) for your study entitled *"The Impact of emotion on Cognition and Thinking in Psychosis"*.

The following documents have been received by the Research Support Office

Document title	Version	Date
Prof Richard Bentall CV	No Version	No date
Informed Consent Form	No Version	08/11/12
Research Proposal	No Version	No date
Participant Information Sheet	V3.2	08/11/12

Please note this does **NOT** constitute final Sponsor approval to allow you to commence recruitment. Sponsor Notification to Proceed will be given when final research ethics, financial and other regulatory requirements have been met. Please see Appendix 1 to this letter for further information and a list of the documents required.

If you have not already applied for regulatory approvals through IRAS you may now do so at <https://www.myresearchproject.org.uk/Home.aspx>.

In order to meet the requirements of the Research Governance Framework 2<sup>nd</sup> Ed 2005, the University requires you to agree to the following Chief Investigator responsibilities: -

1. Inform the Clinical Research Governance Team (CRGT) as soon as possible of any SAE's within the University
2. Provide copies to CRGT of annual progress and safety reports sent to NRES and any other regulatory authorities
3. Comply with the Research Governance Framework 2<sup>nd</sup> Ed 2005 and all relevant legislation, including but not limited to the Data Protection Act 1998 and the Human Tissue Act 2004
4. Inform the University of any amendments to, or changes of status in the study prior to submission to NRES or any other regulatory authorities
5. Maintain the study site file
6. Make available for review any study documentation so requested by the sponsors and regulatory authorities
7. Provide the University with draft publications 30 days prior to submission to the publisher
8. University professional indemnity and clinical trials insurances will apply to the study as appropriate. This is on the assumption that no part of the clinical trial will take place outside of the UK. If you wish to conduct any part of the study in a site outside the UK or you wish to sub-contract any part of the study to a third party specific approvals and consideration of appropriate indemnity would be required.

If you have any queries regarding the sponsorship of the study or the above conditions please do not hesitate to contact the Clinical Research Governance Team on 0151 794 8373 (email [sponsor@liverpool.ac.uk](mailto:sponsor@liverpool.ac.uk)).

Yours sincerely

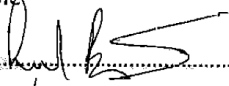


Mrs Lindsay Carter  
Clinical Research Governance Manager, Research Support Office

Cc Head of Institute, Prof Peter Kinderman  
Paulo Sousa

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I agree to the terms and conditions of the University Sponsorship approval for **The Impact of emotion on Cognition and Thinking in Psychosis, UoL000925** and I am aware of my responsibilities under the Research Governance framework. I also agree to provide the Clinical Research Governance Team with the documents listed overleaf when available

CI Signed:  .....

Dated: 6/12/12 .....

Please return a signed copy of this letter to the Research Support Office within 30 days of the date of this letter. Failure to do so may result in Sponsorship being withdrawn.  
Thank you

## **Appendix 1**

In order for the Clinical Research Governance Team to review the clinical research governance elements of the study please provide the following documentation when available;

- NRES Favourable Opinion Letter and all correspondence
- All NRES approved documentation (e.g. Protocol, Participant Information Sheet, Participant Consent Form, GP Letter etc.)
- Copy of trust R&D Approval from Lead NHS Trust
- Sponsor Agreement signed off
- Any other appropriate documentation

Professor Richard Bentall  
Professor of Clinical Psychology  
University of Liverpool  
Institute of Psychology Health & Society  
Waterhouse Block B, B211, 2<sup>nd</sup> Floor  
105 Brownlow Street  
Liverpool L69 3GL

15<sup>th</sup> May 2013

Dear Professor Bentall

**Formal Letter of Approval**

**Project: 2013/5 -The impact of emotion on cognition and thinking in psychosis**

The above research application was disseminated to the Trust's Clinical Business Unit R&D leads for CBU review, and the Trust's Research Governance Committee reviewed the application on the 21<sup>st</sup> March, 2013. The Committee conditionally approved the study subject to a satisfactory response to a number of issues raised within the meeting.

Mr Paulo Sousa kindly responded to the Committee's issues and provided requested documentation. His response was disseminated to the Research Governance Committee with a view to Chair's Action being taken. After consideration, I confirm Chair's Action has been taken to approve the study on behalf of Mersey Care NHS Trust.

Ethical approval has been granted by NRES Committee North West – Preston under reference 118769. Please take this letter as confirmation of Trust R&D approval. Please provide Mr Sousa with a copy of this letter as evidence of research project approval.

Please read the attached 'Information for Researchers – Conditions of Research Governance Approval' leaflet. Please contact the R&D Office should you require any further information, quoting project number 2013/5.

May I wish you every success with his research.

Yours sincerely

  
Mrs Pauline Parker  
R&D Manager

cc. Sponsor contact Liverpool University  
contact: kwilding@liverpool.ac.uk

Chairman: Beatrice Fraenkel

Chief Executive: Joe Rafferty





Research and Effectiveness Office  
1829 Building  
Countess of Chester Health Park  
Chester  
CH2 1HJ

Tel: 01244 650559  
Email: phil.elliott@cwps.nhs.uk

3<sup>rd</sup> April, 2013

Paulo Sousa  
Mental Health and Behavioural Science  
Institute of Psychology, Health and Society  
Waterhouse Building Block B Second Floor,  
1-5 Brownlow Street,  
Liverpool  
L69 3GL

Dear Paulo,

**Letter of Access for Research**

**The impact of emotion on cognition and thinking in psychosis  
(SPEAR number 1211)**

We are satisfied that the research activities that you will undertake in this NHS organisation are commensurate with the activities you undertake for your employer. Your employer is fully responsible for ensuring such checks as are necessary have been carried out. Your employer has confirmed in writing to this NHS organisation that the necessary pre-engagement checks are in place in accordance with the role you plan to carry out in this organisation. This letter confirms your right of access to conduct research through **Cheshire and Wirral Partnership NHS Foundation Trust** for the purpose and on the terms and conditions set out below. This right of access commences on **5<sup>th</sup> April, 2013** and ends on **31<sup>st</sup> March, 2014**, unless terminated earlier in accordance with the clauses below.

You have a right of access to conduct such research as confirmed in writing in the letter of permission for research from this NHS organisation. Please note that you cannot start the research until the Principal Investigator for the research project has received a letter from us giving permission to conduct the project.

You are considered to be a legal visitor to **Cheshire and Wirral Partnership NHS Foundation Trust** premises. You are not entitled to any form of payment or access to other benefits provided by this organisation to employees and this letter does not give rise to any other relationship between you and this NHS organisation, in particular that of an employee.

While undertaking research through **Cheshire and Wirral Partnership NHS Foundation Trust**, you will remain accountable to your employer **University of Liverpool** but you are required to follow the reasonable instructions of your

nominated manager **Dr Pat Mottram, Research and Effectiveness Manager** in this NHS organisation or those given on her/his behalf in relation to the terms of this right of access.

Where any third party claim is made, whether or not legal proceedings are issued, arising out of or in connection with your right of access, you are required to co-operate fully with any investigation by this NHS organisation in connection with any such claim and to give all such assistance as may reasonably be required regarding the conduct of any legal proceedings.

You must act in accordance **Cheshire and Wirral Partnership NHS Foundation Trust** with policies and procedures, which are available to you upon request, and the Research Governance Framework.

You are required to co-operate with **Cheshire and Wirral Partnership NHS Foundation Trust** in discharging its duties under the Health and Safety at Work etc Act 1974 and other health and safety legislation and to take reasonable care for the health and safety of yourself and others while on **Cheshire and Wirral Partnership NHS Foundation Trust** premises. Although you are not a contract holder, you must observe the same standards of care and propriety in dealing with patients, staff, visitors, equipment and premises as is expected of a contract holder and you must act appropriately, responsibly and professionally at all times.

You are required to ensure that all information regarding patients or staff remains secure and *strictly confidential* at all times. You must ensure that you understand and comply with the requirements of the NHS Confidentiality Code of Practice (<http://www.dh.gov.uk/assetRoot/04/06/92/54/04069254.pdf>) and the Data Protection Act 1998. Furthermore you should be aware that under the Act, unauthorised disclosure of information is an offence and such disclosures may lead to prosecution.

**Cheshire and Wirral Partnership NHS Foundation Trust** will not indemnify you against any liability incurred as a result of any breach of confidentiality or breach of the Data Protection Act 1998. Any breach of the Data Protection Act 1998 may result in legal action against you and/or your substantive employer.

You should ensure that, where you are issued with an identity or security card, a bleep number, email or library account, keys or protective clothing, these are returned upon termination of this arrangement. Please also ensure that while on the premises you wear your ID badge at all times, or are able to prove your identity if challenged. Please note that this NHS organisation accepts no responsibility for damage to or loss of personal property.

We may terminate your right to attend at any time either by giving seven days' written notice to you or immediately without any notice if you are in breach of any of the terms or conditions described in this letter or if you commit any act that we reasonably consider to amount to serious misconduct or to be disruptive and/or prejudicial to the interests and/or business of this NHS organisation or if you are convicted of any criminal offence. Where applicable, your substantive employer will initiate your Independent Safeguarding Authority (ISA) registration in-line with the phasing strategy adopted within the NHS and the applicable legislation. Once you are ISA-registered, your employer will continue to monitor your ISA registration status via the on-line ISA service. Should you cease to be ISA-registered, this letter of access is immediately terminated. Your substantive employer will immediately withdraw you from undertaking this or any other regulated activity and you **MUST** stop undertaking any regulated activity.

Your substantive employer is responsible for your conduct during this research project and may in the circumstances described above instigate disciplinary action against you.

If your circumstances change in relation to your health, criminal record, professional registration or ISA registration, or any other aspect that may impact on your suitability to conduct research, or your role in research changes, you must inform the organisation that employs you through its normal procedures. You must also inform your nominated manager in this NHS organisation.

Yours sincerely



**Dr Phil Elliott**  
**Senior Research Facilitator**

## Appendix 2. Consent form



### INFORMED CONSENT FORM

*Title of Research Project:* The impact of emotion on cognition and thinking in psychosis

*Researcher(s):* Prof. Richard Bentall, Dr. William Sellwood and Paulo Sousa

*Please tick box*

1. I confirm that I have read and have understood the information sheet dated ..... for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily by the researcher. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without my rights being affected. ☐
3. I understand that, under the Data Protection Act, I can at any time ask for access to the information I have provided and that I can also request the destruction of that information if I wish. ☐
4. I understand that the study involves the audio recording of interviews and I have been informed that only the research team will be able to listen to these and that I can request the destruction of these recordings if I wish. ☐
5. I agree to take part in the above study. ☐

_____	_____	_____
<i>Participant Name</i>	<i>Date</i>	<i>Signature</i>
_____	_____	_____
<i>Name of person taking consent</i>	<i>Date</i>	<i>Signature</i>
_____	_____	_____
<i>Researcher</i>	<i>Date</i>	<i>Signature</i>

***The contact details of lead Researcher (Principal Investigator) are:***

Professor Richard Bentall  
Institute of Psychology, Health and Society  
Waterhouse Building, Block B, 2nd Floor  
University of Liverpool Brownlow Street  
Liverpool L69 3GL  
0151 795 53 67  
[Richard.Bentall@liverpool.ac.uk](mailto:Richard.Bentall@liverpool.ac.uk)

### Appendix 3. Participant information sheet (clinical)



#### ***Participant Information Sheet***

**Title of Study:** *The impact of emotion on cognition and thinking in psychosis*

**Researchers:** Professor Richard Bentall, Dr William Sellwood and Paulo Sousa

Dear prospective participant,

I'm inviting you to take part in a research study. Before you decide whether you want to take part or not, it is extremely important that you understand why this research is being done and what it will actually involve. Please take time to read the following information sheet carefully and feel free to ask me if there is anything that you do not understand. Please also feel free to discuss your participation with your friends, relatives and care team if you wish. I would like to stress that you do not have to accept this invitation and should only agree to take part if you want to.

#### **1. What is the purpose of the study?**

The purpose of the current study is to look at the impact of emotion on your thoughts and thinking.

#### **2. Why have I been chosen to take part?**

You have been chosen because we are interested in looking at the impact of emotion on the thoughts of people who are experiencing, or may have experienced, mental health difficulties.

#### **3. Do I have to take part?**

Absolutely not, your participation is entirely voluntary. It is up to you to decide if you want to take part and you may discuss your potential participation with your family, friends or your nurse or doctor.

#### **4. What will happen if I take part?**

If you agree to take part, I will arrange for us to meet at a mutually convenient time and location. Each meeting should not take more than 1 hour. During these meetings, I will ask you to tell me about your experiences (e.g. I will ask you to talk about good and bad memories and this can take up to 30 minutes) and to answer 8 questionnaires about your thoughts, feelings and behaviours. During these sessions, breaks can be built if necessary. With your permission, I will audio record the interviews during the meetings so that I can listen to them with more time at a later stage. The recording can be stopped at any time and have words deleted or replaced.

#### **5. Will I be paid if I decide to take part?**

Yes, you'll be paid £20 in the final meeting for your participation and we will reimburse you for any reasonable travelling expenses.

#### **6. Are there any risks in taking part?**

The only potential risk is related to distress that you may experience when we discuss some of your negative memories for example I may ask you to tell me about a time when someone has hurt you and this may bring back a difficult memory.

#### **7. Are there any benefits in taking part?**

The main benefit for you is related to the opportunity to tell us about your thoughts, feelings and behaviours however this and other studies are likely to help improve the care that is provided to mental health patients.

#### **8. What if I am unhappy or if there is a problem?**

If you are unhappy, or if there is a problem, please feel free to let us know by contacting Professor Richard Bentall on 0151 795 5367 ([rpb@liverpool.ac.uk](mailto:rpb@liverpool.ac.uk)) and we will try to

help. If you remain unhappy or have a complaint which you feel you cannot come to us with, then you should contact the Research Governance Officer on 0151 794 8290 ([ethics@liv.ac.uk](mailto:ethics@liv.ac.uk)). When contacting the Research Governance Officer, please provide details of the name or description of the study (so that it can be identified), the researcher involved, and the details of the complaint you wish to make.

#### **9. Will my participation be kept confidential?**

Your participation, as all the information gathered during the meetings, will remain confidential. However, if you disclose information, which indicates that you wish to harm yourself or others, confidentiality will have to be broken and I will have to inform the relevant authority. The questionnaires and tapes (audio-recording) will be kept in a locked cabinet at the Institute of Psychology, Health Society (University of Liverpool).

#### **10. Will my taking part be covered by an insurance scheme?**

Yes, as the study is sponsored by the University of Liverpool and they provide an insurance scheme for researchers.

#### **11. What will happen to the results of the study?**

The results of the study will be submitted for publication with scientific journals. All the participants will be informed of the study results and how to access these publications by letter. No publication deriving from this study will include your personal details or information that can identify you in any way.

#### **12. What will happen if I want to stop taking part?**

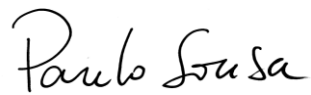
You are free to withdraw from the study at anytime. If you chose to do so, I will ask you if you are happy for me to use the information gathered up to the period of your withdrawal. If not, I will destroy the data and no further use will be made of it. If you decide to withdraw you will still be paid £20.

#### **13. Who can I contact if I have further questions?**

If you have any further questions or things that you would like to see clarified before you decide if you want to take part or not, please free to contact me:

Paulo Sousa  
Institute of Psychology, Health and Society  
Waterhouse Building, Block B, 2nd Floor  
University of Liverpool Brownlow Street  
Liverpool L69 3GL  
0151 795 53 46  
[sousa@liv.ac.uk](mailto:sousa@liv.ac.uk)

Hope to hear from you soon,

A handwritten signature in black ink that reads "Paulo Sousa". The script is cursive and fluid, with the first letter 'P' being large and prominent.



#### Appendix 4. Participant information sheet (comparisons)



##### ***Participant Information Sheet***

**Title of Study:** *The impact of emotion on cognition and thinking in psychosis*

**Researchers:** Professor Richard Bentall, Dr William Sellwood and Paulo Sousa

Dear prospective participant,

I'm inviting you to take part in a research study. Before you decide whether you want to take part or not, it is extremely important that you understand why this research is being done and what it will actually involve. Please take time to read the following information sheet carefully and feel free to ask me if there is anything that you do not understand. Please also feel free to discuss your participation with your friends and relatives if you wish. I would like to stress that you do not have to accept this invitation and should only agree to take part if you want to.

##### **1. What is the purpose of the study?**

The purpose of the current study is to look at the impact of emotion on your thoughts and thinking.

##### **2. Why have I been chosen to take part?**

You have been chosen because we are interested in looking at the impact of emotion on the thoughts of people who are experiencing, or may have experienced, mental health difficulties.

### **3. Do I have to take part?**

Absolutely not, your participation is entirely voluntary. It is up to you to decide if you want to take part and you may discuss your potential participation with your family, friends or your nurse or doctor.

### **4. What will happen if I take part?**

If you agree to take part, I will arrange for us to meet at a mutually convenient time and location. Each meeting should not take more than 1 hour. During these meetings, I will ask you to tell me about your experiences (e.g. I will ask you to talk about good and bad memories and this can take up to 30 minutes) and to answer 8 questionnaires about your thoughts, feelings and behaviours. During these sessions, breaks can be built if necessary. With your permission, I will audio record the interviews during the meetings so that I can listen to them with more time at a later stage. The recording can be stopped at any time and have words deleted or replaced.

### **5. Will I be paid if I decide to take part?**

Yes, you'll be paid £20 in the final meeting for your participation and we will reimburse you for any reasonable travelling expenses.

### **6. Are there any risks in taking part?**

The only potential risk is related to distress that you may experience when we discuss some of your negative memories for example I may ask you to tell me about a time when someone has hurt you and this may bring back a difficult memory.

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The main benefit for you is related to the opportunity to tell us about your thoughts, feelings and behaviours however this and other studies are likely to help improve the care that is provided to mental health patients.

### **8. What if I am unhappy or if there is a problem?**

If you are unhappy, or if there is a problem, please feel free to let us know by contacting Professor Richard Bentall on 0151 795 5367 ([rpb@liverpool.ac.uk](mailto:rpb@liverpool.ac.uk)) and we will try to help. If you remain unhappy or have a complaint which you feel you cannot come to us with, then you should contact the Research Governance Officer on 0151 794 8290 ([ethics@liv.ac.uk](mailto:ethics@liv.ac.uk)). When contacting the Research Governance Officer, please provide details of the name or description of the study (so that it can be identified), the researcher involved, and the details of the complaint you wish to make.

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Your participation, as all the information gathered during the meetings, will remain confidential. However, if you disclose information, which indicates that you wish to harm yourself or others, confidentiality will have to be broken and I will have to inform the relevant authority. The questionnaires and tapes (audio-recording) will be kept in a locked cabinet at the Institute of Psychology, Health Society (University of Liverpool).

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#### **12. What will happen if I want to stop taking part?**

You are free to withdraw from the study at anytime. If you chose to do so, I will ask you if you are happy for me to use the information gathered up to the period of your withdrawal. If not, I will destroy the data and no further use will be made of it. If you decide to withdraw you will still be paid £20.

### **13. Who can I contact if I have further questions?**

If you have any further questions or things that you would like to see clarified before you decide if you want to take part or not, please free to contact me:

Paulo Sousa  
Institute of Psychology, Health and Society  
Waterhouse Building, Block B, 2nd Floor  
University of Liverpool Brownlow Street  
Liverpool L69 3GL  
0151 795 53 46  
[sousa@liv.ac.uk](mailto:sousa@liv.ac.uk)

Hope to hear from you soon,

A handwritten signature in black ink that reads "Paulo Sousa". The script is cursive and fluid, with the first letter 'P' being large and prominent.

## Appendix 5. Positive and Negative Syndrome Scale (PANSS)

Version 4

PANSS – Re-ordered (includes sample prompts)			
a) Patient ID	[ ][ ][ ]	c) Baseline/ EOT/Follow-up (circle)	
b) Assessor's signature:		d) Date	[ ][ ][ ][ ][ ][ ]

### ☐ G1 Somatic concern (physical complaints/beliefs about bodily illness or malfunctions)

- How has your physical health been in the last week?
- Do you ever worry that you have something wrong with your body?
- Do you have a physical illness or disease?
- Does your head or body ever feel strange?
- Or do you have a problem with the way your body has been functioning?
- Has your head or body changed in shape or size?

If answer is YES to any of the above:

- How serious is the problem?
- What is causing the problem?

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	Distinctly concerned about health or somatic issues, evidenced by occasional questions or desire for reassurance
4	Complains about poor health/body malfunction, but no delusional conviction, and over-concern can be allayed by reassurance.
5	Patient expresses numerous or frequent complaints about physical illness or bodily malfunction, or reveals 1 or 2 clear cut delusions involving these themes, but is not preoccupied by them.
6	Patient is preoccupied by one or a few clear-cut delusions about physical or organic malfunction, but affect is not fully immersed in these themes, and thoughts can be diverted by the interviewer with some effort.
7	Numerous and frequently reporting somatic delusions, or a few with catastrophic nature. Which dominate affect and thinking.

### ☐ G2 Anxiety (experiences of nervousness, worry, apprehension, or restlessness)

- Do you find that you worry about things a lot?
  - Have you been feeling nervous/tense/afraid within the last week?
- If YES,
- How anxious have you been feeling on a scale of 1 to 10, with 10 being the most anxious you could ever feel?

If answer is YES to any of the above:

- Are you afraid of something or someone?
- Do you ever get into a state of panic? Or feel shaky/faint/sweaty as a result of feeling anxious?
  - *Definition of panic attack = a feeling of intense fear and anxiety which usually comes on quite suddenly and lasts for a brief amount of time. During an attack, people usually have unpleasant bodily sensations such as: rapid heart beat, breathing very fast, feeling short of breath, chest pains, feeling faint or dizzy, trembling and sweating.*
- Have your worries or nervousness affected your appetite/sleep/ability to work in the last week?

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	Some worry, over-concern or subjective restlessness, but no somatic/behavioural consequences are reported or evident
4	Patient reports distinct symptoms of nervousness, reflected in mild physical manifestations (e.g. fine hand tremors/perspiration)
5	Serious anxiety problems which have significant physical/behavioural consequences (e.g. marked tension, poor concentration, palpitations, impaired sleep)
6	Almost constant fear associated with phobias, marked restlessness or numerous somatic manifestations
7	Life seriously disrupted by anxiety which is present almost constantly, and at times reaches panic proportion or is manifested in actual panic attacks.

**G3 Guilt feelings (self-blame for real or imagined misdeeds in the past)**

- Do you tend to blame yourself for things that have happened?
- Do you feel guilty about something you may have done in the past?
- Do you ever feel like you deserve punishment for something you have done?

If YES,

- What kind of punishment do you deserve?
- What do you deserve punishment for? Is there a particular incident you have in mind?
- Have you had thoughts of harming yourself as one kind of punishment? Have you ever acted on those thoughts?

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	Questioning elicits a vague sense of guilt/self blame for a minor incident, but is clearly not overly concerned
4	Expresses distinct concern over responsibility for a real incident but is not preoccupied by it, and attitude/behaviour are essentially unaffected.
5	Patient expresses strong sense of guilt associated with self-deprecation or the belief that he/she deserves punishment. The guilt feelings may have a delusional basis and may be volunteered spontaneously, may be a source of pre-occupation and or depressed mood, and cannot be allayed readily by the interviewer.
6	Strong ideas of guilt that take on delusional quality – lead to hopelessness and worthlessness. Patient believes he/she deserves harsh sanctions for the misdeeds, and may regard his/her current life situation as such punishment.
7	Patients life dominated by unstable delusions of guilt, for which he/she feels deserving of drastic punishment (e.g. imprisonment, torture, death). There may be associated suicidal thoughts or attribution of others' problems to one's own past misdeeds.

**G6 Depression (feelings of sadness, discouragement, helplessness and pessimism)**

- What has your typical mood been like in the last week?
- Are you mostly happy or sad?
- Have you had periods of feeling sad and hopeless in the last week?

If patient is mostly sad:

- How unhappy have you been feeling on a scale of 1 to 10, with 10 being the most unhappy you could feel?
- When do you feel the saddest? How long do these feelings last?
- Do you sometimes cry? How often?
- Has your low mood affected your appetite/sleep/ability to work?
- Do you have less or nearly no interest that you used to in your leisure/social activities or hobbies or things you used to enjoy?
- Have you had thoughts of harming yourself?

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	Expresses some sadness or discouragement only on questioning, but there is no evidence of depression in general attitude of demeanour.
4	Distinct feelings of sadness/hopelessness, which may be spontaneously divulged, but depressed mood minimally affects behaviour/social functioning. Can usually be cheered up.
5	Distinct depressed mood associated with obvious sadness, pessimism, loss of social interest, psychomotor retardation, and some interference in appetite or sleep. Patient cannot easily be cheered up.
6	Markedly depressed mood, misery, hopelessness, worthlessness, occasional crying. Major interference with appetite and/or sleep as well as normal motor and social functions. Signs of possible self-neglect.
7	Depressive feelings seriously interfere in most major functions. Frequent crying, pronounced somatic symptoms, impaired concentration, self neglect, social disinterest, possible depressive or nihilistic delusions. Possible suicidal thoughts/actions.

**G12** Lack of judgement and insight (impaired awareness/understanding of one's psychiatric condition and life situation. Denial of the need for treatment, inability to recognise psychiatric symptoms, unrealistic short-term and long-term planning)

- Do you generally feel that you are in need of help and treatment from people such as Dr XXX (patients doctor)
- Do you feel you have a psychiatric illness or do you feel you have had one in the past?  
If YES
- What is it?
- How serious do you feel it is on a scale of 1 to 10 (10 being the most serious it could be)
  - Where do you see yourself/what would you hope to be doing in 1 years time/5 years time?

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	Recognises psychiatric disorder but underestimates seriousness, implications for treatment or the importance of taking measures to avoid relapse. Future planning may be poorly conceived
4	Vague/shallow recognition of illness. Fluctuations in acknowledgement of being ill or little awareness of major symptoms that are present such as delusions, disorganised thinking, suspiciousness and social withdrawal. May rationalise treatment to relieve lesser symptoms e.g. anxiety, poor sleep etc.
5	Acknowledge past but not present disorder. If challenged, may concede the presence of some unrelated or insignificant symptoms which tend to be explained away by gross misinterpretation or delusional thinking. Need for treatment is unrecognised.
6	Denies ever having a psychiatric disorder, Patient disavows the presence of any psychiatric symptoms in the past or present, and denies the need for treatment/hospitalisation.
7	Emphatic denial of past and present illness with current hospitalisation/treatment given a delusional interpretation (eg. As a punishment for misdeeds, or persecution by tormentors) The patient may refuse to cooperate with therapists, medication or other aspects of treatment.

**P1** Delusions (beliefs that are unfounded, unrealistic, and idiosyncratic/peculiar)

*Delusions of reference*

- Do you feel at times that others make references or say things with a double meaning?
- Do you see messages for yourself in the newspaper or on TV?
- Do you occasionally feel that some events or incidents have a special meaning particularly for you?

*Delusional misinterpretation*

- Do you occasionally see a secret message in the way objects are arranged or in their labelling or colour or in the way things happen?

*Quotation of ideas*

- Do you find that something you have previously thought or discussed is quoted on TV or in the newspapers, or used in some other way to indicate a reference to you?

*Familiar people impersonated*

- Do you feel that the appearance of any people you know well has changed in ways that suggest that someone might be impersonating them?

*Delusions of persecution*

- Does anyone seem to be trying to harm you?  
If YES are they particularly singling you out?
  - How do you experience this?
  - Does there seem to be a plot or a conspiracy behind it?
    - How do you recognise it?

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	1 or 2 delusions that are vague, uncrystallised and not tenaciously held. Delusions do not interfere with thinking, social relations or behaviour
4	Presence of either a kaleidoscopic array of poorly formed, unstable delusions or a few well formed delusions that occasionally interfere with patients thinking, social relations or behaviour.
5	Numerous well formed delusions that are tenaciously held and occasionally interfere with patients thinking, social relations or behaviour
6	Stable set of delusions that clearly interfere with patients thinking, social relations and behaviour
7	Highly systemised or very numerous stable delusions, that dominate major facets of patients life. Often results in inappropriate/irresponsible action that may jeopardise safety of patient or others.

**P5** **Grandiosity** (exaggerated self-opinion and unrealistic convictions of superiority, including delusions of extraordinary abilities, wealth, knowledge, fame, power and moral righteousness)

- How do you feel you compare to the average person? Better or worse?
- Do you have talents/abilities/special or unusual powers that most people don't have?
  - For example, do you ever feel you read another person's mind?
- Do you consider yourself wealthy? Famous? Have you ever appeared on television, radio, movies or stage?
- Do you rate higher in terms of your moral standards?
  - Does this make you special in some respect?
- Do you have a special mission in life?
  - How did this come about?
- Are you a religious person?
  - What is your relationship with god?
  - Are you closer to god than others are?

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	Some expansiveness or boastfulness is evident, but without clear-cut grandiose delusions.
4	Feels Distinctly and unrealistically superior to others. Some poorly formed delusions about special status/abilities may be present but not acted upon.
5	Clear-cut delusions concerning remarkable abilities/status /power that influence patients attitude but not behaviour
6	Clear cut delusions of remarkable superiority involving more than 1 parameter (wealth, fame, knowledge) are expressed, notably influence interactions, and may be acted upon
7	Thinking, interactions and behaviour are dominated by multiple delusions of amazing ability/wealth/knowledge/fame/power/moral stature which may take on a bizarre quality.

**P6** **Suspiciousness/Persecution** (unrealistic/exaggerated ideas of persecution are shown, as reflected in guardedness, a distrustful attitude, suspicious hypervigilance, or delusions that others mean one harm

- How do you feel you get along with other people?
- Do you like other people? Dislike people?
  - If patient dislikes people :
    - Do you get particularly annoyed with people?
    - Afraid of people? Why?
- Do you feel most people like you? Dislike you? Why?
- Do you trust most people you know?
  - Are there some whom you distrust? Who? Why?
- Do you ever feel some people talk about you behind your back?
  - What do you think they say? Why?
- Do you ever feel some people spy on you/plot against you/attempt to harm you/attempt to kill you?
  - What is the evidence for this?
  - Who is behind all this?
  - Why does it happen?

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	Presents a guarded or openly distrustful attitude, but thoughts/interactions/behaviour are minimally affected.
4	Distrustfulness is clearly evident, intrudes on interview and his/her behaviour, but there is no evidence of persecutory delusions. Or loosely formed persecutory delusions which do not seem to affect patients' attitude/interpersonal relations.
5	Patient shows marked distrustfulness, leading to major disruptions in interpersonal relations. Or clear cut delusions that have limited impact on his/her interpersonal relations and behaviour.
6	Clear cut pervasive delusions of persecution which may be systematised and that significantly interfere in patients interpersonal relations
7	A network of systemised persecutory delusions dominates the patients thinking, social relations and behaviour



P7

**Hostility** (verbal & non-verbal expressions of anger and resentment, including sarcasm, passive-aggressive behaviour, verbal abuse and assaultiveness)

- **How have you been getting along with people lately? (family, co-workers etc)**  
If patient hasn't been getting on well with people – why?
- **Have you been irritable or grumpy lately?**  
If YES, does this lead to arguments with others even about minor issues, which normally wouldn't bother you?
  - Were you ever so irritable that you would shout out at people or start arguments or fights?

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	Indirect or restrained communication of anger (e.g. sarcasm, disrespect, hostile expressions or occasional irritability)
4	Patient presents an overtly hostile attitude showing frequent irritability and direct expression of anger or resentment
5	Highly irritable and occasionally verbally abusive or threatening
6	Uncooperativeness and verbal abuse or threats notably influence the interview and seriously impact upon patients social relations. Patient may be violent and destructive but not physically assaultive towards others
7	Marked anger results in extreme uncooperativeness precluding other interactions, or in episodes of physical assault towards others.

P3

**Hallucinatory behaviour** (verbal report or behaviour indicate perceptions that are not generated by external stimuli. May be auditory, visual, olfactory or somatic)

- **Do you ever have strange experiences/hear strange noises or sometimes hear things that others don't hear?**
- **Do you sometimes receive personal communications from the radio or television?**
- **Can you sometimes hear your thoughts aloud in your head? Do they sound like voices?**  
If patient hears voices:
  - How many are there?
  - Do they speak to you, comment about you, or speak to each other?
  - What do the voices say?
  - Are they good or bad voices?
  - Are you afraid of them?
  - Do the voices tell you what to do? Give you direct orders?
    - Do you obey the voices' commands? Must you?
- **Do ordinary things ever appear strange or distorted or do you ever have visions or see things others don't?**  
If YES,
  - How often?
  - How clear are these visions?
  - Do the visions occur together with the voices or separately?
- **Do you ever smell things that others don't?**
- **Do you ever get strange sensations from within your body or feel something strange inside you?**  
  
If patient reports voices or visions, explore further with:
  - What do you make of these voices / visions etc...?
  - How did they come about?
  - Are they a problem?

Version 4

1	The definition doesn't apply
2	Questionable pathology; may be at the upper extreme of normal limits
3	1 or 2 clearly formed but infrequent hallucinations or a number of vague abnormal perceptions which do not result in distortions of thinking or behaviour.
4	Hallucinations occur frequently but not continuously, and the patients thinking and behaviour are affected only to a minor extent.
5	Hallucinations are frequent, may involve more than one sensory modality, and tend to distort thinking and/or disrupt behaviour. Patient may have a delusional interpretation of these experiences and respond to them emotionally and, on occasion, verbally as well.
6	Hallucinations are present almost continuously, causing major disruption of thinking and behaviour. Patient treats these as real perceptions, and functioning is impeded by frequent emotional and verbal responses to them.
7	Patients is almost totally preoccupied with hallucinations, which virtually dominate thinking and behaviour. Hallucinations are provided a rigid delusional interpretation and provoke verbal and behavioural responses, including obedience to command hallucinations.

**G13 Disturbance of volition** (disturbance in wilful initiation, sustenance, and control of one's thoughts, behaviour, movements and speech.

- Do you find it difficult to make decisions in your day to day life?
  - If YES, has this occurred in the last week?
  - Example?
- Do you find your behaviour is sometimes aimless and disconnected, so that your daily routine is chaotic, because you are unable to plan your actions properly?
  - If answer YES to any of these, explore further, ask for an example/why do you think this is etc.

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	Some evidence of indecisiveness in conversation and thinking that may impede verbal and cognitive processes to a minor extent
4	The patient is often ambivalent and shows clear difficulty reaching decisions. Conversation may be marred by thinking alteration, and consequently, his or her verbal and cognitive functioning are clearly impaired.
5	Disturbance of volition interferes in behaviour and thinking. Pronounced indecision that impedes the initiation and continuation of social and motor activities, and which may be evidenced in halting speech
6	Execution of simple automatic motor functions (e.g. dressing/grooming) is interfered with, and speech is markedly affected.
7	Almost complete failure of volition is manifested by severe inhibition of movement and speech.

**G10 Disorientation** (lack of awareness of one's relationship to one's surroundings, including persons, places, and time that may be due to confusion or withdrawal)

- Do you know what day it is today?
- Month?
- Year?
- Season?
- Date?
- Where we are?

1	The definition doesn't apply
2	Questionable pathology – patient may be upper extreme of normal limits
3	General orientation is adequate but patient may have difficult with specifics, for example knows their location but not street address, knows hospital staff names but not their function, knows month but confuses day of the week. There may be narrowing of interest evidenced by familiarity with immediate but not extended milieu (ie identifies the staff but not Prime Minister etc).
4	Only partial success in recognising persons, places and time. For example, patient knows they are in a hospital but it's name, knows name of primary therapist but not many other direct care worker, knows year but not sure of month.
5	Considerable failure in recognising persons, places and time, for example has only vague notion of their whereabouts, and unfamiliar with most people in their milieu. May know year but not month, day or season.
6	Marked failure in recognising persons, places and time. (no knowledge of whereabouts, confuses date, can only name 1 or 2 individuals in current life.
7	Complete disorientation with regard to persons, places and time. Gross confusion or total ignorance about location, the current year, and even the most familiar people, such as parents, spouse, therapist etc.

Version 4

☐ **N5** **Difficulty in abstract thinking** (impairment in abstract-symbolic thinking, as demonstrated by difficulty in classification, forming generalisations, and moving beyond concrete or egocentric thinking in problem solving tasks)

(See appendix I for list)

1	The definition doesn't apply
2	Questionable – patient may be upper extreme of normal limits
3	Tends to give literal or personalised interpretations to the more difficult proverbs, and some problems with concepts that are fairly abstract or remotely related
4	Often utilises concrete mode. Difficulty with most proverbs and some categories. Tends to be distracted by functional aspects and salient features.
5	Patient deals primarily in concrete mode, exhibiting difficulty with most proverbs and many categories.
6	Unable to grasp abstract meaning of proverbs or figurative expressions and can formulate classifications for only the most simple of similarities. Thinking is either vacuous or locked into functional aspects, salient features, and idiosyncratic interpretations.
7	Only uses concrete thinking modes. No comprehension of proverbs, common metaphors or similes and simple categories. Even salient and functional attributes do not serve as a basis for classification. This rating may apply to those who cannot interact even minimally with the interviewer due to marked cognitive impairment.

☐ **G9** **Unusual thought content** (thinking is characterised by strange or bizarre ideas, ranging from those that are remote/atypical to those that are distorted and absurd)

Basis for rating: Thought content expressed during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Thought content is somewhat peculiar or idiosyncratic, or familiar ideas are framed in an odd context.
4	Ideas are frequently distorted and occasionally seem quite bizarre.
5	Patient expresses many strange and fantastic thoughts (eg. Being adopted son of a king, being an escapee from death row) or some which are patently absurd (eg. Having hundreds of children, receiving radio messages from space via a tooth filling).
6	Patient expresses many illogical or absurd ideas or some which have a distinctly bizarre quality (eg having 3 heads, being a visitor from another planet).
7	Thinking is replete with absurd, bizarre and grotesque ideas.

☐ **N3** **Poor rapport** (lack of interpersonal empathy, openness in conversation, and a sense of closeness, interest, or involvement with the interviewer. This is evidenced by interpersonal distancing and reduced verbal and nonverbal communication)

Interpersonal behaviour during the course of interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Conversation is characterised by a stilted, strained, or artificial tone. It may lack emotional depth or tend to remain on an impersonal, intellectual plane.
4	Patient typically is aloof, with interpersonal distance quite evident. Patient may answer questions mechanically, act bored, or express disinterest.
5	Disinvolvement is obvious and clearly impedes the productivity of the interview. Patient may tend to avoid eye or face contact.
6	Patient is highly indifferent, with marked interpersonal distance. Answers are perfunctory, and there is little nonverbal evidence of involvement. Eye and face contact are frequently avoided.
7	Patient is totally uninvolved with the interviewer. Patient appears to be completely indifferent and consistently avoids verbal and nonverbal interactions during the interview.

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Version 4

**N2 Emotional withdrawal** (lack of interest in, involvement with and affective commitment to life events)

Observation of interpersonal behaviour during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Usually lacks initiative and occasionally may show deficient interest in surrounding events.
4	Patient is generally distanced emotionally from the milieu and its challenges but, with encouragement, can be engaged.
5	Patient is clearly deattached emotionally from persons and events in the milieu, resisting all efforts at engagement. Patient appears distant, docile, and purposeless but can be involved in communication at least briefly and tends to personal needs, sometimes with assistance.
6	Marked deficiency of interest and emotional commitment results in limited conversation with others and frequent neglect of personal functions, for which the patient requires supervision.
7	Patient is almost totally withdrawn, uncommunicative, and neglectful of personal needs as a result of profound lack of interest and emotional commitment.

**N4 Passive/apathetic social withdrawal** (diminished interest and initiative in social interactions due to passivity, apathy, anergy or avolition leading to reduced interpersonal involvements and neglect of daily living activities). Reports from others only.

How do you spend your time these days? Do you prefer to be alone?

Do you join in on activities with others?

(if not) Why not?

Do you have many friends?

(If no) do you have any friends?

Do you have any close friends?

How often do you see them?

(if not) Why not?

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Shows occasional interest in social activities but poor initiative. Usually engages with others only when approached first by them.
4	Passively goes along with most social activities but in a disinterested or mechanical way. Tends to recede into the background.
5	Passively participates in only a minority of activities and shows virtually no interest or initiative. Generally spends little time with others.
6	Tends to be apathetic and isolated, participating very rarely in social activities and occasionally neglecting personal needs. Has very few spontaneous social contacts.
7	Profoundly apathetic, socially isolated, and personally neglectful.

**G16 Active social avoidance** (diminished social involvement associated with unwarranted fear, hostility or distrust). Reports from others only.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Patient seems ill at ease in the presence of others and prefers to spend time alone, although she/he participates in social functions when required.
4	The patient begrudgingly attends all or most social activities but may need to be persuaded or may terminate prematurely on account of anxiety, suspiciousness, or hostility.
5	Patient fearfully and angrily keeps away from many social interactions despite others' efforts to engage them. Tends to spend unstructured time alone.
6	Patient participates in very few social activities because of fear, hostility or distrust. When approached, the patient shows a strong tendency to break off interactions, and generally tends to isolate themselves.
7	Patient cannot be engaged in social activities because of pronounced fears, hostility, or persecutory delusions. Avoids all interactions and remains isolated from others.

**P2 Conceptual disorganisation** (Disorganised process of thinking characterised by disruption of goal-directed sequencing, eg., circumstantiality, tangentiality, loose associations, non-sequiturs, thought block or gross illogicality).

Basis for rating: Cognitive verbal processes observed during the course of interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Thinking is circumstantial, tangential, or paralogical. There is some difficulty in directing thoughts toward a goal, and some loosening of associations may be evidenced under pressure.
4	Able to focus thoughts when communications are brief and structured, but becomes loose or irrelevant when dealing with more complex communications or when under minimal pressure.
5	Generally has difficulty in organising thoughts, as evidenced by frequent irrelevancies, disconnectedness, or loosening of associations, even when not under pressure.
6	Thinking is seriously derailed and internally inconsistent, resulting in gross irrelevancies and disruption of thought processes, which can occur almost constantly.
7	Thoughts are disrupted to the point where the patient is incoherent. There is marked loosening of associations, which results in total failure of communication, eg., 'word salad', or mutism.

**G7 Motor retardation** (Reduction in motor activity reflected by the slowing or lessening of movements and speech, diminished responsiveness to stimuli, and reduced body tone).

Basis for rating: manifestations during the course of the interview

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Slight but noticeable diminution in rate of movements and speech; patient may be somewhat unproductive in conversation and gestures.
4	Patient is clearly slow in movements, and speech may be characterised by poor productivity, including long response latency, extended pauses, or slow pace.
5	A marked reduction in motor activity renders communication highly unproductive or delimits functioning in social and occupational situations. Patient can usually be found sitting or lying down.
6	Movements are extremely slow, resulting in a minimum of activity and speech. Essentially the day is spent idly or lying down.
7	Patient is almost completely immobile and virtually unresponsive to external stimuli.

**N6 Lack of spontaneity and flow of conversation** (reduction in the normal flow of communication associated with apathy, avolition, defensiveness, or cognitive deficit. This is manifested by diminished fluidity and productivity of the verbal-interactive process.

Rating based on cognitive-verbal processes observed during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Conversation shows little initiative. Patients answers tend to be brief and unembellished, requiring direct and leading questions by the interviewer.
4	Conversation lacks free flow and appears uneven or halting. Leading questions are frequently needed to elicit adequate responses and proceed with conversation.
5	Patient shows a marked lack of spontaneity and openness, replying to the interviewers questions with only one or two brief sentences.
6	Patient's responses are limited mainly to a few words or short phrases intended to avoid or curtail communication (eg 'I don't know', 'I'm not at liberty to say'). Conversation is seriously impaired as a result, and the interview is highly unproductive.
7	Verbal output is restricted to, at most, an occasional utterance, making conversation not possible.

**N7 Stereotyped thinking** (decreased fluidity, spontaneity, and flexibility of thinking, as evidenced in rigid, repetitious, or barren thought content)

Rated on cognitive verbal processes observed during the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Some rigidity shown in attitudes or beliefs. Patient may refuse to consider alternative positions or have difficulty in shifting from one idea to another.
4	Conversation revolves around a recurrent theme, resulting in difficulty in shifting to a new topic.
5	Thinking is rigid and repetitious to the point that, despite the interviewers efforts, conversation is limited to only two or three dominating topics.
6	Uncontrolled repetition of demands, statements, ideas, or questions which severely impairs conversation.
7	Thinking, behaviour, and conversation are dominated by constant repetition of fixed ideas or limited phrases, leading to gross rigidity, inappropriateness and restrictiveness of patients communication.

**N1 Blunted affect** (diminished emotional responsiveness characterised by a reduction in facial expression, modulation of feelings, and communicative gestures).

Observed manifestations of affective tone and emotional responsiveness during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Changes in facial expression and communicative gestures seem to be stilted, forced, artificial, or lacking in modulation.
4	Reduced range of facial expression and few expressive gestures result in a dull appearance.
5	Affect is generally 'flat', with only occasional changes in facial expression and paucity of communicative gestures.
6	Marked flatness and deficiency of emotions exhibited most of the time. There may be unmodulated extreme affective discharges, such as excitement, rage, or inappropriate uncontrolled laughter.
7	Changes in facial expression and evidence of communicative gestures are virtually absent. Patient seems constantly to show a barren or 'wooden' expression.

Version 4

☐ **P4 Excitement** (hyperactivity is reflected in accelerated motor behaviour, heightened responsivity to stimuli, hypervigilance, or excessive mood lability.)

Rating based upon behavioural manifestations during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Tends to be slightly agitated, hypervigilant or mildly overaroused throughout the interview, but without distinct episodes of excitement or marked mood lability. Speech may be slightly pressured.
4	Agitation or overarousal is clearly evident throughout the interview, affecting speech and general mobility or episodic outbursts occur sporadically.
5	Significant hyperactivity or frequent outbursts of motor activity are observed, making it difficult for the patient to sit longer than several minutes at any given time.
6	Marked excitement dominates the interview, delimits attention and to some extent affects personal functions such as eating or sleeping.
7	Marked excitement seriously interferes in eating and sleeping and makes interpersonal interactions virtually impossible. Acceleration of speech and motor activity may result in incoherence and exhaustion.

☐ **G5 Mannerisms and posturing** (unnatural movements or posture are shown as characterised by an awkward, stilted, disorganised, or bizarre appearance).

Ratings based on the observation of physical manifestations during the course of interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Slight awkwardness in movements or minor rigidity of posture.
4	Movements are notably awkward or disjointed, or an unnatural posture is maintained for brief periods.
5	Occasional bizarre rituals or contorted posture are observed, or an abnormal position is sustained for extended periods.
6	Frequent repetition of bizarre rituals, mannerisms, or stereotyped movements, or a contorted posture is sustained for extended periods.
7	Functioning is seriously impaired by virtually constant involvement in ritualistic, manneristic, or stereotyped movements or by an unnatural fixed posture which is maintained most of the time.

☐ **G14 Poor impulse control** (there is disordered regulation and control when acting on inner urges, resulting in sudden, unmodulated, arbitrary, or misdirected discharge of tension and emotions without concern about the consequences).

Basis for rating: Behaviour during the course of the interview or else otherwise reported.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Patient tends to be easily angered and frustrated when facing stress or denied gratification but rarely acts on impulse.
4	Patient get angered and verbally aggressive with minimal provocation. May be occasionally threatening, destructive, or have one or two episodes involving physical confrontation or a minor brawl.
5	Patient exhibits repeated impulsive episodes involving verbal abuse, destruction of property, or physical threats. There may be one or two episodes involving serious assault, for which the patient requires isolation, physical restraint, or sedation.
6	Patient frequently is impulsively aggressive, threatening, demanding, and destructive, without any apparent consideration of consequences. Shows assaultive behaviour and may also be sexually offensive and possibly respond behaviourally to hallucinatory commands.
7	Patient exhibits homicidal attacks, sexual assaults, repeated brutality, or self-destructive behaviour. Requires constant direct supervision or external constraints because of inability to control dangerous impulses.

☐

Version 4

**G4 Tension** (There are overt physical manifestations of fear, anxiety, and agitation, such as stiffness, tremors, profuse sweating, and restlessness).

Based upon verbal report attesting to anxiety, and thereupon the severity of physical manifestations of tension observed during the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Posture and movements indicate slight apprehensiveness, such as minor rigidity, occasional restlessness, shifting of position, or rapid hand tremor.
4	A clearly nervous appearance emerges from various manifestations, such as fidgety behaviour, obvious hand tremor, excessive perspiration, or nervous mannerisms.
5	Pronounced tension is evidenced by numerous manifestations, such as nervous shaking, profuse sweating, and restlessness, but conduct in the interview is not significantly affected.
6	Pronounced tension to the point that interpersonal interactions are disrupted. The patient, for example, may be constantly fidgeting, unable to sit still for long, or show hyperventilation.
7	Marked tension is manifested by signs of panic or gross motor acceleration, such as rapid restless pacing an inability to remain seated for longer than a minute, which makes sustained conversation not possible.

**G8 Uncooperativeness** (active refusal to comply with the will of significant others, including the interviewer, hospital staff, or family, perhaps associated with distrust, defensiveness, stubbornness, negativism, rejection of authority, hostility, or belligerence.

Basis for rating: Interpersonal behaviour observed during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Complies with an attitude of resentment, impatience, or sarcasm. May inoffensively object to sensitive probing during the interview.
4	Occasional outright refusal to comply with normal social demands, such as making own bed, scheduled appointments etc. The patient may project a hostile, defensive, or negative attitude but usually can be worked with.
5	Patient is frequently noncompliant with the demands of his/her milieu and may be characterised by others as an 'outcast' or having a serious 'attitude problem'. Uncooperativeness is reflected in obvious defensiveness or irritability with the interviewer and may be unwilling to address many questions.
6	Patient is highly uncooperative, negativistic, and possibly also belligerent. Refuses to comply with most social demands and may be unwilling to initiate or conclude the full interview.
7	Active resistance seriously impact on virtually all major areas of functioning. Patient may refuse to join in any social activities, tend to personal hygiene, converse with family or staff, and participate even briefly in an interview.

**G11 Poor attention** (poor focussed alertness is manifested by poor concentration, distractibility from internal and external stimuli, and difficulty in harnessing, sustaining, or shifting focus to new stimuli.)

Basis for rating: Manifestations during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Limited concentration evidenced by occasional vulnerability to distraction or faltering attention toward the end of the interview.
4	Conversation is affected by the tendency to be easily distracted, difficulty in long sustaining concentration on a given topic, or problem shifting attention on to new topics.
5	Conversation is seriously hampered by poor concentration, distractibility, and difficulty in shifting focus appropriately.
6	Patients attention can be harnesses for only brief moments or with great effort, due to marked distraction by internal or external stimuli.
7	Attention is so disrupted that even brief conversation is not possible.



☐ **G15 Preoccupation** (there is an absorption with internally generated thoughts and feelings or with autistic experiences to the detriment of reality orientation and adaptive behaviour.

Interpersonal behaviour reported during the course of the interview.

1	The definition doesn't apply
2	Questionable pathology; patient may be upper extreme of normal limits
3	Excessive involvement with personal needs or problems, such as that conversation veers back to ego-centric themes and there is diminished concern exhibited toward others.
4	Patients occasionally appears self-absorbed, as if daydreaming or involved with internal experiences, which interferes with communication to a minor extent.
5	Patient often appears to be engaged in autistic experiences, as evidenced by behaviours that significantly intrude on social and communicational functions, such as the presence of a vacant stare, muttering or talking to oneself, or involvement with stereotyped motor patterns.
6	Marked preoccupation with autistic experiences, which seriously delimits concentration, ability to converse, and orientation to the milieu. The patients frequently may be observed smiling, laughing, muttering, talking or shouting to oneself.
7	Gross absorption with autistic experiences, which profoundly affects all major realms of behaviour. The patient constantly may be responding verbally and behaviourally to hallucinations and show little awareness of other people or the external milieu.

## **Appendix 6. Psychosis Screening Questionnaire (PSQ)**

### **HYPOMANIA**

PROBE: *Over the past year, have there been times when you felt very happy indeed without a break for days on end?*

If yes,

- *Was there an obvious reason for this?*
- *Did your relatives or friends think it was strange or complain about it?*

### **THOUGHT INSERTION**

PROBE: *Over the past year, have you ever felt that your thoughts were directly interfered with or controlled by some outside force or person?*

If yes,

- *Did this come about in a way that many people would find hard to believe, for instance, through telepathy?*

### **PARANOIA**

PROBE: *Over the past year, have there been times when you felt that people were against you?*

If yes,

- *Have there been times when you felt that people were deliberately acting to harm you or your interests?*
- *Have there been times when you felt that a group of people were plotting to cause you serious harm or injury?*

### **STRANGE EXPERIENCES**

PROBE: *Over the past year, have there been times when you felt that something strange was going on?*

If yes,

- *Did you feel it was so strange that other people would find it very hard to believe?*

### **HALLUCINATIONS**

PROBE: *Over the past year, have there been times when you heard or saw things that other people couldn't?*

If yes,

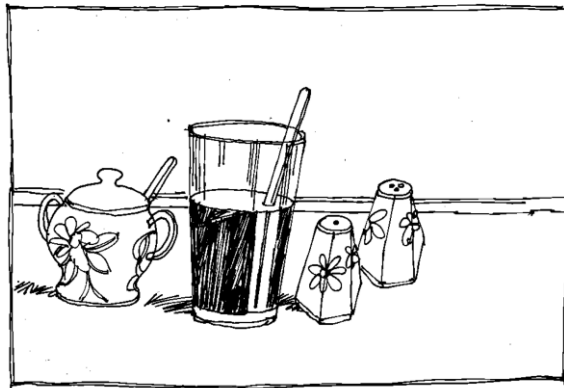
- *Did you at any time hear voices saying quite a few words or sentences when there was no one around that might account for it?*

**Appendix 7. QUICK test (cards and words)**

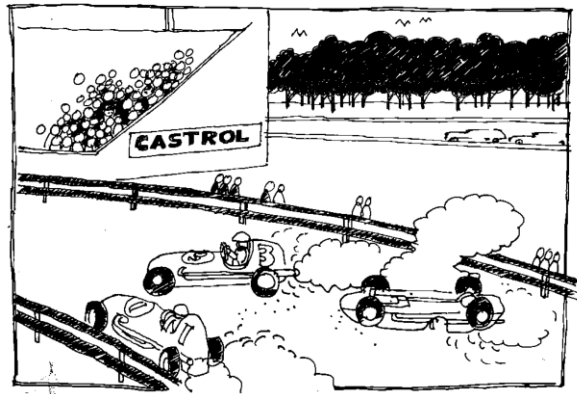
**Card 1**



**Card 2**



Card 3



Card 4



## Words

belt dancing traffic whistle fence	5	graceful fluid solution discipline bleachers	30
drink wreck music medicine gun	10	crystallized turntable saccharin immature cordiality	35
pepper racing salt woman sugar	15	velocity decisive laceration foliage imperative	40
track school partner couples rail	20	intimacy concoction conviviality chevrons condiment	45
respectful betting daring stadium pedestrian	25	cacophony miscible imbibe amicable pungent	50

## **Appendix 8. Salient and non-salient interviews**

### **Interview questions**

Each question is preceded by "tell me about..."

#### Questions for Emotionally Salient Interview

1. A close relative or friend who died.
2. When you felt most unloved by your parents.
3. The most awful thing that someone has done to you.
4. When someone you cared about was very ill.
5. When someone who you loved let you down.
6. A time when someone has hurt you.
7. When someone you loved abandoned you.
8. The times when you have felt that life is not worth living.
9. The people in your family.
10. The things that make you feel most sad.
11. A funeral that you attended.
12. A major disagreement you had with someone you loved.
13. An important relationship, which ended unhappily.
14. The arguments that you had in your family.
15. The times when you've felt very embarrassed.

#### Reserve Questions for Emotionally Salient Interview

1. The things you worry about most.
2. The happiest times in your life.
3. The times when your family were proud of you.
4. The things that make you most happy.
5. A time when someone was especially kind to you.
6. The places where you have felt most happy.
7. The times when you've felt most useful in your life.
8. A person you admire a lot.

#### Questions for Non-Emotionally Salient Interview

1. A building in Manchester.
2. Travelling on public transport.
3. How you feel about cleaning.
4. How you feel about current fashion in clothes.
5. A sporting event that you remember.
6. A famous sports-person.
7. How you feel about going to the public library.
8. Nurses' pay.

9. How you feel about listening to the radio.
10. The recent advances in space.
11. What you think about Tony Blair.
12. What you feel about politics.
13. How you feel about big cities.
14. A film star who you admire.
15. How you feel about cooking.

#### Reserve Questions for Non-Emotionally Salient Interview

1. How you feel about going to the cinema.
2. Your favourite foods.
3. Your favourite TV programme.
4. An incident that has been in the news recently.
5. How you feel about shopping.
6. A hobby which you are most interested in.

## Appendix 9. TLC scoring sheet

### Scale for the assessment of thought, language and communication (TLC)

ID: \_\_\_\_\_ Interview: Neutral/Salient

TLC Items	0	1	2	3	4
Poverty of speech					
Poverty of content of speech					
Pressure of speech					
Distractible speech					
Tangentiality					
Derailment					
Incoherence					
Illogicality					
Clanging					
Neologisms					
Word approximations					
Circumstantiality					
Loss of goal					
Perseveration					
Echolalia					
Blocking					
Stilted speech					
Self-reference					
Phonemic paraphasia					
Semantic paraphasia					
<b>Global rating</b>					



## **Appendix 10.** Source monitoring (instructions, task, recognition sheet and example card)

### **Instructions**

Before beginning the task, show participants the practice statement and the two types of cards that may follow it. Tell the participants:

“I’m going to show you a series of statements. Each statement ends with a blank and I want you to think of a word that completes the statement. Don’t state your answer out loud. Just nod your head to indicate that you have an answer prepared. I will then show you one of two cards. If I show you the card that says “Answer”, I want you to state your answer out loud. If I show you a card with a new statement on it, I want you to go ahead and to prepare an answer for the new statement. Nod your head to indicate when you have an answer. Do you have any questions?”



If participants ask if they have to keep remembering their old answers, tell them no, they should just go ahead and prepare an answer for the next statement.

Researchers should record all of participants’ verbal responses. Also record any instances in which the participants accidentally give a say response to a think statement.

Immediately after participants have completed the statements they should complete the source recognition sheet. Read instructions on that sheet to participant. Check to make sure that participants have completed all the items of the recognition sheet.

## Source Monitoring Task (version a)

This is the item order for both versions of the task. The letters “S” and “T” indicate whether a statement requires a say or a think response. This is the type of responses that would be given for version A of this measure. **Version b reverses the type of response required.**

1. There are seven days in a \_\_\_\_\_. (S) week
2. This shape  is called \_\_\_\_\_. (S) triangle
3. In the UK, we celebrate Halloween on the 31<sup>st</sup> of \_\_\_\_\_. (T) October
4. The Queen’s first name is \_\_\_\_\_. (S) Elizabeth
5. A lift goes up and \_\_\_\_\_. (T) down
6. In the UK, five quid means five \_\_\_\_\_. (S) pounds
7. The sun rises in the East and sets in the \_\_\_\_\_. (T) West
8. Between meals, a person may eat a \_\_\_\_\_. (T) snack
9. The first month of the year is \_\_\_\_\_. (S) January
10. A fiver is equivalent to \_\_\_\_\_ pounds. (T) five
11. This shape  is called a \_\_\_\_\_. (T) circle
12. The first meal of the day is called \_\_\_\_\_. (S) breakfast
13. The current prime minister in the UK is called David \_\_\_\_\_. (T) Cameron
14. When the winter comes, the birds fly \_\_\_\_\_. (S) South
15. The opposite of left is \_\_\_\_\_. (S) right
16. A day is divided into twenty-four \_\_\_\_\_. (T) hours

## Recognition sheet

**Instructions:** The words below are words that you may have said or thought to complete the previous sentences or the words may be new words. For each word, mark whether it was a word that you said, a word that you thought, or a word that is new. Write an “S” after the words that you remember saying, write an “T” after the words you remember thinking, and write an “N” after words that you think are new.

Answer all items. If you are unsure of the correct answer, guess.

1. January \_\_\_\_\_
2. Pence \_\_\_\_\_
3. Elizabeth \_\_\_\_\_
4. Down \_\_\_\_\_
5. Cameron \_\_\_\_\_
6. Lunch \_\_\_\_\_
7. Hours \_\_\_\_\_
8. South \_\_\_\_\_
9. October \_\_\_\_\_
10. Breakfast \_\_\_\_\_
11. Quid \_\_\_\_\_
12. Right \_\_\_\_\_

1. North \_\_\_\_\_
2. Forward \_\_\_\_\_
3. Snack \_\_\_\_\_
4. Minutes \_\_\_\_\_
5. Five \_\_\_\_\_
6. Triangle \_\_\_\_\_
7. West \_\_\_\_\_
8. Circle \_\_\_\_\_
9. Blair \_\_\_\_\_
10. December \_\_\_\_\_
11. Week \_\_\_\_\_
12. Square \_\_\_\_\_

There are seven days in a

\_\_\_\_\_.

Answer

## Appendix 11. Positive and Negative Affect Scale (PANAS)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. Indicate to what extent you feel this way **right now**, that is, at the present moment.

1	2	3	4	5
Very slightly or not at all	A little	Moderately	Quite a bit	Extremely

Interested____	Irritable____
Distressed____	Alert____
Excited____	Ashamed____
Upset____	Inspired____
Strong____	Nervous____
Guilty____	Determined____
Scared____	Attentive____
Hostile____	Jittery____
Enthusiastic____	Active____
Proud____	Afraid____

## **Appendix 12.** Lubben's Social Network Scale (LSNS)

**FAMILY:** Considering the people to whom you are related by birth, marriage, adoption, etc....

1. How many relatives do you see or hear from at least once a month?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

2. How often do you see or hear from relative with whom you have the most contact?

0 = less than monthly 1 = monthly 2 = few times a month 3 = weekly 4 = few times a week 5 = daily

3. How many relatives do you feel at ease with that you can talk about private matters?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

4. How many relatives do you feel close to such that you could call on them for help?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

5. When one of your relatives has an important decision to make, how often do they talk to you about it?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

6. How often is one of your relatives available for you to talk to when you have an important decision to make?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

**NEIGHBOURS:** Considering those people who live in your neighbourhood...

7. How many of your neighbours do you see or hear from at least once a month?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

8. How often do you see or hear from the neighbour with whom you have the most contact?

0 = less than monthly 1 = monthly 2 = few times a month 3 = weekly 4 = few times a week 5 = daily

9. How many neighbours do you feel at ease with that you can talk about private matters?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

10. How many neighbours do you feel close to such that you could call on them for help?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

11. When one of your neighbours has an important decision to make, how often do they talk to you about it?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

12. How often is one of your neighbours available for you to talk to when you have an important decision to make?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

**FRIENDSHIPS:** Considering your friends who do not live in your neighbourhood...

13. How many of your friends do you see or hear from at least once a month?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

14. How often do you see or hear from the friend with whom you have the most contact?

0 = less than monthly 1 = monthly 2 = few times a month 3 = weekly 4 = few times a week 5 = daily

15. How many friends do you feel at ease with that you can talk about private matters?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

16. How many friends do you feel close to such that you could call on them for help?

0 = none 1 = one 2 = two 3 = three or four 4 = five thru eight 5 = nine or more

17. When one of your friends has an important decision to make, how often do they talk to you about it?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

18. How often is one of your friends available for you to talk to when you have an important decision to make?

0 = never 1 = seldom 2 = sometimes 3 = often 4 = very often 5 = always

*LSNS-R total score is an equally weighted sum of these twelve items. Scores range from 0 to 90*



### **Appendix 13. Varieties of Inner Speech Questionnaire (VISQ)**

- 1) I think to myself in words using brief phrases and single words rather than full sentences
- 2) When I am talking to myself about things in my mind, it is like I am going back and forward asking myself questions and then answering them
- 3) I hear the voice of another person in my head. For example, when I have done something foolish
- 4) I hear my mother's voice criticising me in my mind.
- 5) I experience the voices of other people asking me questions in my head.
- 6) I hear other people's voices nagging me in my head.
- 7) My thinking in words is more like a dialog with myself, rather than my own thoughts in a monolog
- 8) I think to myself in words using full sentences
- 9) My thinking to myself in words is like shorthand notes, rather than full, proper, grammatical English
- 10) I think in inner speech about what I have done, and whether it was right or not
- 11) When I am talking to myself about things in my mind, it is like I am having a conversation with myself
- 12) I talk silently to myself telling myself to do things
- 13) I hear other people's actual voices in my head, saying things that they have never said to me before
- 14) I talk back and forward to myself in my mind about things
- 15) My thinking in words is shortened compared to my normal out-loud speech. For example, rather than saying to myself things like 'I need to go to the shops', I will just say 'shops' to myself in my head
- 16) If I were to write down my thoughts on paper, they would read like a normal grammatical sentence
- 17) I hear other people's actual voices in my head, saying things that they actually once said to me
- 18) I talk silently to myself telling myself not to do things

- 19) I evaluate my behaviour using my inner speech. For example I say to myself, “that was good” or “that was stupid”

Scale ranges from 1 (*certainly does not apply to me*) to 7 (*certainly applies to me*).

#### Appendix 14. Self-concept Clarity Scale (SCCS)

1. My beliefs about myself often conflict with one another \*
2. On one day I might have one opinion of myself and on another day I might have a different opinion \*
3. I spend a lot of time wondering about what kind of person I really am \*
4. Sometimes I feel that I am not really the person that I appear to be \*
5. When I think about the kind of person I have been in the past, I'm not sure what I was really like \*
6. I seldom experience conflict between the different aspects of my personality
7. Sometimes I think I know other people better than I know myself \*
8. My beliefs about myself seem to change very frequently \*
9. If I were asked to describe my personality, my description might end up being different from one day to another day \*
10. Even if I wanted to, I don't think I could tell someone what I'm really like \*
11. In general, I have a clear sense of who I am and what I am
12. It is often hard for me to make up my mind about things because I don't really know what I want \*

Scale ranges from 1 (*strongly disagree*) to 5 (*strongly agree*).

\* Indicates reverse-keyed item.